Original Communications*

PRESIDENTIAL ADDRESS

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IT IS needless to tell you of my appreciation of the great honor which you have extended to me in electing me your presiding officer, the greatest honor that it has ever been my fortune to attain. For your confidence I thank you and trust that my administration of the office may justify it.

This Association has an enviable record, but it has yet many functions to perform for the betterment of American obstetrics, gynecology, and abdominal surgery. In my humble opinion the future development of the dual specialties must be the result of a firmer fusion between these two branches of medicine, for the basic pathology of the diseases which affect pelvic organs in women is founded upon the pathology of the traumatic lesions of childbirth and their consequent infections, and no surgeon who has not had training in obstetric pathology by autopsy study of the gross pathologic picture, supplemented by a study of the reparative and protective microscopic changes which take place in these tissues, can fully appreciate the resources of defense which Nature summons to her aid in the repair and arrest of these conditions.

Present-day surgery has accepted physiologic pathology as its guide for interference, and, if gynecic surgery is to keep abreast with the advances in other special lines, the men who go into this specialty must be equipped with this knowledge, as it pertains to the anatomic structures which constitute pelvic organs in women. It is, therefore, important that this Association place itself on record in favor of the combined Chair in the teaching of obstetrics and gynecology to our undergraduates; for the obstetrician must be a surgeon, likewise the gynecologist, a diagnostician.

That our obstetric defects are many is shown by the comparative study of the mortality statistics of any well-organized clinic, contrasted with the study of similar statistics as to the cause of death recorded in a metropolitan or rural health bureau. It is deplorable in this century, which shines with accomplishments in preventive medicine that any analysis of the deaths incident to childbirth show that 43 per cent are the direct or indirect result of infection; 26 per cent or more are due to the toxemias of pregnancy, while fully 10 per cent are directly the result of operative causes. Such a mortality from preventable causes does not obtain at the present time in any other branch of medicine, neither does it obtain in England, France, Germany, or Scandinavia. Let us, therefore, seek to find an explanation for this lamentable fact.

Careful investigation of existing conditions which I have made, shows that the didactic teaching of obstetrics in all Class A medical schools is uniformly good, but is not given the time commensurate with its importance. The student, under our present system, has such a very limited clinical training—so inadequate—that he is not in a position to recognize slight defects in mechanism, and their effect on the physiologic course of labor; neither is he properly equipped to do the common obstetric operations. The average student in the best clinics sees not more than fifty cases of labor, and probably delivers less than twenty. With the ordinary incidence of abnormalities, dystocias, and accidents, this means the possible chance of his seeing one breech delivery, two low forceps, one version, one cesarean section and, possibly, watching the treatment of one patient with a preeclampsia.

No man with such a paucity of surgical knowledge and experience would attempt to assume the responsibility of an abdominal operation, a thoracic paracentisis, or a mastoid drainage. Yet the graduate assumes, and the public accepts him as competent to handle, any obstetric delivery which in every sense is a surgical procedure. Even a midwife, trained abroad, has had ten times the clinical experience of our graduates in medicine, for every foreign midwife must have delivered at least two hundred cases, including versions and extractions, before being admitted to practice: even then she is subject to medical supervision and is responsible to the State for her errors, while our graduate, after passing the State Board examination and receiving his license, is controlled only by his conscience. Notwithstanding this, the Committee on Curriculum of the American Association of Medical Colleges and of the American Medical Association, apparently discounting the fact that obstetrics as it is taught in this country today is more poorly practiced than any other branch of medicine, are recommending a five-week block system of clinical obstetrics which will further curtail the clinical experience of the average medical graduate.

Obstetrics, medicine, and surgery should have an equal division of hours in the clinical year if we are to better the training of our graduates and give better service to the public. The importance of aseptic detail during the conduct of the first stage of labor should be so impressed upon the student that when obstetric operations are indicated and must be done, they will not be followed with the morbidity and mortality so common in present-day practice.

Routine manikin drill should be the coincident accompaniment of every clinic labor. By this method we can demonstrate the mechanism which is actually taking place in the case at hand, and so clarify the significance of many of the malpositions and their resulting dystocias. No textbook description, however lucid, can compare with visual demonstration or leave a like impression on the mind of the student. Furthermore, the usual custom of not calling a student until the woman is about to be delivered, or is in the second stage of labor, fails to teach him the important and essential point in every labor; namely, that dilatation takes time and pains, and that there are indications for and certain basic conditions necessary to accomplish safe delivery.

More emphasis should be placed on the fact that full dilatation of the cervix is a necessity before attempting any form of infravaginal delivery, and that this is accomplished by pains, time, and the physiologic pressure of the hydrostatic bag, less perfectly by the presenting part, and, finally, that manual or instrumental dilatation can never simulate the perfection of nature's physiologic mechanism.

Each student should be drilled in making abdominal, rectal, and vaginal examinations, in order to determine the presentation, position, steps of mechanism and to check the progress of the labor, as well as to recognize the degree of cervical dilatation.

Many men leave college with these facts driven home, only to read an article from the pen of some well-known teacher, calling attention to a method of shortening normal labor by operative measures. This Association cannot control the expression of the individual opinions of trained specialists, in whose hands radical procedures are relatively safe, but it can father the dissemination of rational practice for the rank and file. There should be a minimum standard of technic in obstetrics, just as there is a minimum standard in surgery.

The necessity for an endowment fund was conceived by your Secretary, to whose wisdom and energy we owe so much. This Association is a national institution with purposes and functions to perform that will outlive all of us as individuals, and I need not tell you that it takes money as well as brains to accomplish these. Even with the modest fund which is being established, we shall be able to spread our special knowledge to the general practitioner, stimulate research in the individual, and possibly establish a yearly fellowship for the

better training of eligible candidates. As the public will be the benefactor, it is from it—through you—that we may expect additions to this fund.

Another one of the responsibilities of this Association is not only to use its influence for the better teaching of obstetrics and gynecology to both the graduate in practice and the undergraduate, by encouraging the establishment of special teaching hospitals, but to endorse the lengthening of residence of the student in these services, so that he may become familiar with the diagnosis and management of obstetrics and gynecologic emergencies.

Another need of our Association is an editorial committee. Our Secretary is already taxed beyond his physical powers, yet he acts as editor of our transactions, for which he receives a nominal stipend and a deal of criticism. At each session there are from twenty to thirty contributions to our medical literature; many are suitable for journal publication, while others are of such a radical nature that the suggestions contained therein should be subjected to further trial by the trained expert before publication in the ordinary medical channels. These, of course, should be published as the author's individual opinion in our transactions, but should not go out to the profession with the visé of our Association until further proof of their excellence is submitted. An editorial committee selected from the teachers of our specialties in the universities should function with power in these matters, and in this way our Association would blaze the way toward better standards of teaching.

The present trend of obstetrics and gynecology is toward a better understanding of the underlying physiology, anatomy, and pathology of the lesions that we, as specialists, have to treat. Empiricism is fast becoming a thing of the past; we are more and more appreciative of both the interrelation and independence prevailing between gynecologic and obstetric lesions and general medicine, likewise of the significance of focal infections.

The uterus and its adnexa have been found to have less significance in the causation of symptoms than was formerly attributed to these organs. On the other hand, preoperative study and exact diagnosis are receiving more and more attention, while operative measures are falling into the class of preventive procedures rather than curative ones. Ablations are becoming less common as the significance of the interbalance of the endocrine glands is becoming better understood. All of these facts show that the obstetrician and gynecologist must be primarily a physician, trained in internal medicine with a firm grounding in his knowledge of anatomy, physiology, pathology, and bacteriology.

Studies of infection have shown that special bacteria have particular routes of invasion, a selectivity of tissue, and that they produce

definite and characteristic pathology. The permanence and extent of this pathology will depend on the type of bacterium and on the degree of tissue resistance. This latter varies with the condition and type of the individual. Thus, we find the gonococcus in certain locations with a limited life history, for the tissues affected show a reaction and power of regeneration capable of combating the invader; such a patient is left with but a limited permanent pathology. Furthermore, we have learned that the so-called exacerbations in pelvic inflammatory disease are, in reality, reinfections which originate from uncured foci, such as are found in the urethral and cervical glands.

On the other hand, puerperal and postabortal infections due to streptococci and staphylococci have a resulting pathology that is always more or less permanent, likewise the life history of the invading coccus is always questionable and varies from a few months to many years; hence, the significance of having a detailed history of the attack and a knowledge of the bacteriology, when determining the time for operative treatment. It has been proved beyond question that the uterus, its adnexa, and the surrounding parametrium and peritoneum are capable of combating a pure gonorrheal invasion, and that the results of such an infection can be so perfectly corrected by nature that pregnancy can and does occur; yet latent infections may remain quiescent in the urethral glands and in the glands of the uterine cervix for long periods of time and still retain their infectivity.

Admitting these clinical facts, radical extirpations in this form of infection, except when the invasion has been one of mixed origin, are procedures to be postponed; while, on the other hand, infections of puerperal or operative origin frequently show a wider degree of permanent tissue destruction, as well as a greater tendency to affect remote organs through extension, by the lymphatics and blood stream which seem less able than the uterus to cope with these virulent types of cocci. Furthermore, pathogenic bacteria having a greater longevity, become buried in an exudate or sealed in a wound, only to become reactivated by surgical trauma. All studies point toward the conservation of tissue and the capability of nature's resistance.

Notwithstanding the excellent work of Trendelenburg, Vineberg, and J. F. Baldwin, who, by ligation of the pelvic veins or panhysterectomy, aim to limit or extirpate the local infection, most authorities are convinced from the operating table picture, supplemented by autopsy and microscopic studies, that nature is doing just what we attempt to do in a better and more positive way, and that surgery, no matter by whom it is done, breaks down nature's barrier and spreads the infection.

Stimulation of the pituitary glands or the intramuscular use of pituitary extract in conjunction with repeated small transfusions of whole blood are now accepted as rational aids in the stimulation of

nature's supporting processes. Surgical procedure in the presence of streptococcic infection is limited in obstetrics and gynecology, as it is in surgery, to the incision and drainage of pus collection.

The repair of childbirth trauma is an accepted principle, but the time for such repair is still a question of debate. Theoretically, the immediate suture of such a lesion is ideal and should give satisfactory results; practically, however, owing to the severe tissue trauma and the usual accompanying edema, there is interference with the expected perfect union. In all the extensive injuries occurring in primiparae, our best results have been obtained by waiting for twenty-four hours, for even in this short time the edema more or less disappears, the injuries can be better exposed, and their extent more readily appreciated; hence, coaptation is more perfect and suture constriction may be avoided. The most aseptic technic, of course, is imperative.

Multiparae who have been subjects of previous obstetric injuries may have these lacerations repaired during the lying-in period. Here the intermediate procedure has been followed with excellent success. Our best results have been obtained by operating at the end of the first week, at which time, if there has been an afebrile convalescence, the cervix, anterior wall, and pelvic floor may be restored with reasonable expectation of success. Birth injuries left uncared for lead to chronic invalidism from prolapse, displacement, menstrual disturbances and predisposition to local infection, local irritation, or faulty drainage which, in turn, favor the development of cancer of the cervix.

Taking up a consideration of the present status of uterine cancer we find, according to Hoffman's statistics for the year 1923, that out of every 100,000 persons dying from cancer in the United States, 57,397 were females and of these more than 40 per cent had the disease located in the genital organs or in the breasts. Soper, Hoffman, and others state that the incidence of cancer in the United States is increasing, and support this statement with detailed statistical tables.

These facts are discouraging and would appear to justify the pessimism of the public and some of the profession were it not that the great mass of experimental and clinical work which is being carried on by research students in this country and abroad has resulted in establishing certain incontestable facts which are of the utmost clinical value. It is to these that I wish to call your attention. While we must admit that our knowledge relative to the etiology and prevention of this malady is still defective, experimental and clinical study has resulted in giving us the most complete understanding of the pathology, incidence, routes of invasion, and clinical behavior of all forms of cancer. This is particularly true of cancer of the cervix and uterine body, for growths in this location are accessible to attack. The methods of diagnosis are positive and accurate, and the curative

treatment is effective, if the disease is detected in its earliest stage. Certainly these should be encouraging facts for both the profession and the public.

To combat any given disease, three basic factors must be dealt with: (1) the essential or primary cause, (2) the exciting or secondary cause, and (3) the cure. In cancer, the first factor is still unknown, though there are many theories to explain its origin. Graves, in a recent review of the cancer problem in gynecology, states that "of the various theories that seek to explain the neoplastic change of the cell, that of 'cell autonomy' is perhaps the most convenient as a working basis, for this theory assumes that normal cells grow under certain restraints which include: (1) tension of the surrounding tissues; (2) proper nutrition; (3) the necessity for function, and (4) the purposeful control of the organism, and that tumor cells which are emancipated from these growth restraints are merely exhibiting their normal capacity for growth, and become lawless." This so-called cell autonomy is so inclusive that in a general way it covers most of the well-known hypotheses as to the origin of cancer. Of these the dedifferentiation theory of Blair-Bell seems the most tenable and convincing, for it is supported not only by morphologic, chemical, and physiologic evidence, but by incontestable and demonstrable clinical results which have been achieved in the treatment of human cancer. Bell sees "in malignant neoplasia a reversion of the somatic cell to the earliest embryonic type which is represented by the chorionic epithelium. This dedifferentiation of the normal somatic cells may supervene on the common precancerous conditions produced by irritative factors, such as mechanical trauma, chronic inflammation, radiation, heat, and irritating chemicals. In the precancerous state there appears to be an oxygen starvation of the cell; this necessitates that the cell must then, either recover, or die, or undergo dedifferentiation, assuming the trophoblastic form in order to provide itself with sufficient nutriment."

The analogy of malignant cells to chorionic tissue is shown morphologically by the tendency to syncytial arrangement and by their power to invade blood vessels. Chemically, both tissues when compared with the normal show a high phosphatide-cholesterol ratio, which is an absolute requisite for cell membrane permeability and rapid growth.

Physiologically, the dedifferentiation theory is supported by the investigations of Warburg, who has proved that malignant cells obtain their energy by glycolysis in contrast to the oxidation method of nutrition of the normal tissues. In further support of Warburg's observations, Blair-Bell, of Liverpool, and Murphy, of the Rockefeller Institute, have shown that chorionic tissue behaves in an identical manner.

Toxicologically, both malignant and chorionic tissues show the same reaction; namely, that of coagulation necrosis to lead, while clinically,

this theory is supported by the observation that abortions are specifically frequent among lead workers, contrasted with the fact that persons suffering from chronic lead poisoning never develop cancer. While the acceptance of this theory as to the essential cause is still debatable, certain secondary causes particularly those of chronic irritation and chronic inflammation have long been accepted as precursors of cancer.

The work of Gye, who believes that the stimulating virus of cancer is due to an invisible, filterable bacterium, deserves attention for opening up new fields of investigation that are already leading toward tenable grounds.

In contrast to these speculative theories certain clinical observations regarding the effect of chronic irritation and chronic inflammation have been recorded for many years. These are best illustrated in the neglected lacerations and chronic erosions of the cervix which are more or less constant precursors of cervical epithelioma. These clinical observations have been further supported by experimental studies of the production of cancer in rats and rabbits by chronic irritation.

The incidence of obstetric traumas preceding cancer of the cervix is estimated at from 90 to 98 per cent. In the analysis by Graves of 538 cases of cancer of the cervix there was an incidence of 91 per cent; all of these women had borne children or had had one or more miscarriages. Contrasted with this is a study of the hospital records of 4,815 cervical repairs, which included the operations of trachelorrhaphy, amputation, and cauterization; follow-up records found only *seven* cases that later developed cancer, and in three of these malignant changes were already present in the tissue removed at the time of the primary operation. Such statistics as these, coming as they do from a reliable source, direct our attention to the importance of the repair of cervical lesions as a cancer prophylactic.

In the study of body cancers, Graves found a large proportion of senile atresias which produce a tangible irritative process by causing the retention of uterine secretions, chemically changed and exerting pressure and irritation for long periods on the senile epithelium of low differentiation, with its tendency to heap up into papillomatous masses. So constant was this observation that we must admit the possibility of gynatresia acting as an etiologic factor in corporeal cancer.

The diagnosis is accurate and positive if reliance is placed on routine microscopic study of all biopsy and curetted material. There are no physical signs which are pathognomonic of incipient cancer. In cancer of the cervix only Group I cases are amenable to total extirpation or cell destruction by irradiation; hence, it is apparent that the early detection is the keynote of success, for every failure to make the diagnosis in the incipient stage costs a human life.

From this brief review we can make the following deductions: (1) that cervical cancer may be in great part prevented by the proper attention to obstetric trauma and to chronic cervical infections, and the incidence of corporeal cancer may be diminished by establishing proper drainage in the gynatresias; (2) that early detection is imperative and that routine microscopic studies of all tissues removed at trachelorrhaphy, tracheloplasty, or amputation and of all curettings will discover malignant neoplasia at a time when radical surgery or cell destruction is possible, for, unless this extirpation or destruction is complete, surgery not only shortens the woman's life but makes her life more miserable.

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THE FLUCTUATION IN BLOOD SUGAR DURING ECLAMPSIA AND ITS RELATION TO THE CONVULSIONS

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EVIDENCE is now available, as the result of an arduous series of investigations over a period of several years, which indicates that the opinions generally held regarding certain important blood chemistry changes in eclampsia are partially incorrect.

As stated in our preliminary report,[1] we are now prepared to furnish proof by means of these studies, (1) that a disturbance in carbohydrate metabolism in eclampsia actually exists, (2) that contrary to the general opinion, hyperglycemia is not characteristic of eclampsia, but (3) that eclamptic convulsions are directly related to and probably the result of hypoglycemic levels during the course of this disease.

It is obvious that if our observations are correct, they must have a definite and direct bearing on both the etiology and the treatment of eclampsia.

THEORETIC RELATIONSHIP BETWEEN DISTURBANCE OF CARBOHYDRATE METABOLISM AND TOXEMIAS OF PREGNANCY

Hofbauer[2] was the first to adduce evidence of the glycogen depletion of the liver which is "common" to pregnancy, and he even designated the histologic changes resulting from this process as "the liver of pregnancy." Furthermore, his analyses of the glycogen content of the livers of eclamptics showed a relationship between the degree of glycogen depletion of the liver and the pathologic degenerative changes seen in the tissue of this organ.

As long ago as 1920 one of us (Titus[3]) postulated the theory that a profound disturbance in carbohydrate metabolism resulting from an actual deficiency in carbohydrates seemed to be the underlying factor in the development of pregnancy toxemias. Duncan and Harding[4] had advanced a similar theory shortly before, but the explanation of the source of this deficiency as conceived independently in our clinic differed somewhat from theirs and was briefly as follows: "The liver is the carbohydrate storing organ of the body, its cells being filled with glycogen, and a carbohydrate deficiency in the maternal organism causes a glycogen depletion of the liver. Such a deficiency of carbohydrates during pregnancy may be of twofold origin: (1) There is an unexpected demand for glycogen on the part of the fetus, as shown by Slemons[5] and others, and to a lesser degree by the rapid hypertrophy of the uterus, this being a relative deficiency, and (2) an actual deficiency augmented in the presence of nausea and vomiting, from lessened carbohydrate intake as the result of an improperly balanced diet. All degrees of variation in this are possible during pregnancy."

It was reasoned that depletion of the liver cells of their stored glycogen might well be the starting point for the development of a pregnancy toxemia, and it was shown by further work of our group[6] that this also had a direct relationship to the pathologic lesions of the liver which are seen in these toxemias, thus corroborating certain of Hofbauer's earlier findings.

THERAPEUTIC APPLICATIONS OF CARBOHYDRATE DEFICIENCY THEORY

Our earlier studies dealt chiefly with hyperemesis, because this toxicosis of early pregnancy is so common that large numbers of cases are always available for study. Cases of eclampsia were, of course, included.

Based on this theoretical deficiency and disturbance in metabolism of carbohydrates, we developed a definite line of treatment, and one important feature of this was the recommendation made here for the first time that the intravenous administration of hypertonic glucose solution be utilized for all toxemias of pregnancy. This treatment, begun experimentally and suggested by Litchfield's[7] work with glucose in pneumonias, has since been developed and extended both by us and by others to the point where it has now become quite generally accepted and successfully used, not only in obstetrics but also in surgery.

Williams[8] in the last edition of his textbook, as well as in later publications, accepted and approved the suggestion of intravenous glucose therapy because of its liver-sparing effect but stated that in eclampsia there is no evidence of a carbohydrate insufficiency as we had suggested.

It seemed inconceivable, however, that our reasoning along such definite physiologic lines could be so faulty, especially when the clinical results in this treatment of even severe grades of hyperemesis were so favorable. Glucose therapy seemed to be a most reasonable thing for eclampsia if for no other reason than that the enormous consumption of energy incidental to the convulsions required this fuel to replace the depleted stores. The results of this therapy promptly proved to be equally satisfactory to those seen in hyperemesis. An intravenous injection of a strongly hypertonic glucose solution has a striking effect in controlling both the severity and the number of the convulsions as well as in lowering blood pressure and stimulating diuresis.

CURRENT OPINION ON BLOOD CHEMISTRY IN ECLAMPSIA

It has been reiterated by many authorities that an increase in blood sugar (hyperglycemia) is characteristic of eclampsia. Various investigators have made the general statement that blood chemistry studies have been disappointing because the only changes seen in eclampsia are an increase in sugar, and a lesser increase in uric and lactic acids, none of which seemed to throw any light on the etiology of the disease.

In his latest publication on the subject of eclampsia Williams[9] says that "there is a marked increase in the uric acid content (of the blood) followed by equally striking changes in the amount of sugar and lactic acid. . . . . The changes noted in eclampsia . . . . do not in any way bear out the original supposition that it is associated with nitrogenous retention, but indicate a profound disturbance of metabolism." He gives as the usual average of blood sugar during eclampsia a hypercontent of from 120 to 185 mg. per 100 c.c.

Benthin[10] was among the first to say that in eclampsia the blood sugar is nearly always raised above the upper border of normal, concluding that this increase is an effect of the muscular activity during the convulsions. Widen[11] found an intermittent hyperglycemia as a characteristic symptom of eclampsia and believed that the prognosis was improved in the presence of increased blood sugar. Slemons[12] refers to the hyperglycemia following eclamptic convulsions and for lack of evidence he too discards the nitrogenous retention theory of the disease. Plass[13] agrees with this latter and also says "thus far, no definite metabolic changes have been associated with the actual convulsive seizures."

Stander and Duncan[14] even go so far as to make the suggestion that because of the constancy of hyperglycemia in eclampsia one should take this for granted when laboratory facilities are lacking, and administer insulin even without the precaution of carrying out blood-sugar readings.

CONTRADICTORY OBSERVATIONS ON BLOOD SUGAR IN ECLAMPSIA

In connection with some glycemia-curve estimations in eight eclamptic women reported in an earlier paper by our group,[6] it was noticed that only four of them showed an initial blood sugar above 110 mg. per 100 c.c., and one of the other four was as low as 77 mg. Obata and Hayashi[15] have found the blood-sugar levels in eclamptics approx-

imating those of normal women, although a few of their cases showed some increase. They quote both Bergsma and Moritani as having been unable to find any difference between the blood-sugar levels in normal women and eclamptics.

Recently, Levy[16] of New Orleans has commented as follows: "findings of an increased blood-sugar content (in eclampsia) are directly contrary to mine of a consistently lowered one."

It seemed apparent, therefore, that hyperglycemia is not as constant a feature of eclampsia as was commonly assumed. Parenthetically it may be said that lactic acid is merely a by-product of muscle-work[17] and that experimental muscular contractions are followed by a definite ratio of lactic-acid increase in both muscles and blood stream up to the point of muscular exhaustion. We are not concerned with this at present, however, more than to offer it as an explanation of what has been considered another constant of eclamptic blood chemistry; namely, increase in lactic acid.

DEVELOPMENT OF GLYCEMIA CURVE STUDIES DURING ECLAMPSIA

The discovery of insulin was an enormous stimulant to research in carbohydrate metabolism. It was soon shown that insulin overdosage produces a lowering of blood sugar with a simultaneous depletion of the glycogen of the liver[18] and that at certain hypoglycemic levels severe convulsions occur which could promptly be relieved by the administration of glucose or other carbohydrates.

Our earlier attempts to explain the success of the empiric carbohydrate treatment of pregnancy toxemias on the theory that these intoxications develop only in the presence of a carbohydrate deficiency had previously carried us over a course of reasoning almost parallel in pregnancy toxemia to these new developments in this other field. We were still faced, however, with the glaring inconsistency that eclampsia was apparently accompanied by increased rather than lowered blood-sugar values.

Recalling the lowered blood-sugar readings which we and others had reported in occasional cases of eclampsia, and also that hyperglycemia of varying degree usually follows any sudden muscular exertion, we conceived the idea that there might be fluctuations in the blood sugar during eclampsia.

If our theories were fundamentally correct as we believed, it was logical to expect that blood-sugar values would be low before a convulsion, and that following this tremendous muscular upheaval, they would rise to hyperglycemic levels.

The actual attempts at carrying out an investigation of these ideas proved to have many technical difficulties, and at the outset of this work a number of cases were totally wasted, so far as profitable study was concerned, in the effort to perfect our details of technic. It was simple enough to reason that blood taken just before a con-

vulsion might show a vastly different sugar level from that taken shortly after, but no one could foretell when a convulsion would occur and therefore when to take such a desirable specimen.

In order to be certain of obtaining such specimens, it was necessary to arrange to take a series of blood samples at stipulated intervals, hoping to be fortunate enough to secure some of them just before convulsive seizures occurred. This often involved hours of tedious and fruitless work, but by serving in relays, we were able in a number of instances to get precisely what we sought.

Another difficulty lay in the matter of treatment. It was quite important from our standpoint that during the time these specimens were being taken the patient should receive no treatment. Consequently the time which could be allotted to these studies was often necessarily shortened by the patients' requirements, since we had no intention of endangering their lives.

INITIAL BLOOD-SUGAR LEVELS IN OUR SERIES OF ECLAMPTICS

Despite the fact that practically all of these patients had been given at least one dose of morphine before being sent to the hospital (according to Stander[19] this increases blood sugar), only four of the thirteen patients studied showed an initial sugar reading above 110 mg. per 100 c.c. of blood. Four were found at average or normal levels (two at 100, one at 105 mg. and one at 110 mg. respectively) while the remaining five were all below 90, one even being at the low level of 55 mg.

Quite contrary, therefore, to the usual opinions, we found normal and lower than normal values predominating.

FLUCTUATIONS IN BLOOD SUGAR DURING ECLAMPSIA, AND THEIR SIGNIFICANCE

As will be seen by the charts presented herewith, it was soon evident that our lines of reasoning had been correct. By taking frequent blood-sugar readings during an attack of eclampsia, wide fluctuations in the values were readily demonstrated, these variations being encountered in surprisingly short intervals of time. Such differences as 50 to 80 mg. or more of sugar were common and repeated occurrences in time intervals of only a few minutes (110 mg. change in fifteen minutes time in one extreme instance).

After noting these striking fluctuations in eclampsia, serial readings of blood-sugar (fasting) values were taken in normal pregnant women near term for control. It will be seen by the graphs in Chart I that these values are maintained at constant levels over a period of several hours, and in no way resemble those of the eclamptic cases. It is planned to extend these normal studies which apparently have not been done before, and to include with them patients having pre-eclampsia and other less acute toxicoses of late pregnancy.

From a study of the curves of our eclampsia cases it is now possible to state as the general rule that eclamptic convulsions are preceded by definite and sudden drops in blood-sugar values which may vary all the way from 10 or 15 mg. to as much as 80 mg. Exceptionally such decreases in our curves were not followed by convulsions, but the rule was so definite that we believe a convulsion was threatened at each of these lowered points.

We had expected to find a sharp rise in blood sugar following each convulsion, and over a considerable period of this research our efforts were bent mainly toward obtaining specimens directly before a convulsion or just as it began, and within a minute or two after the seizure. It was in this way that a number of cases were practically lost for study because the most important findings representative of the entire case were to be developed from the more constant blood-sugar readings and the curve plotted therefrom. This, or the similar technical mistake of taking only occasional specimens might cause
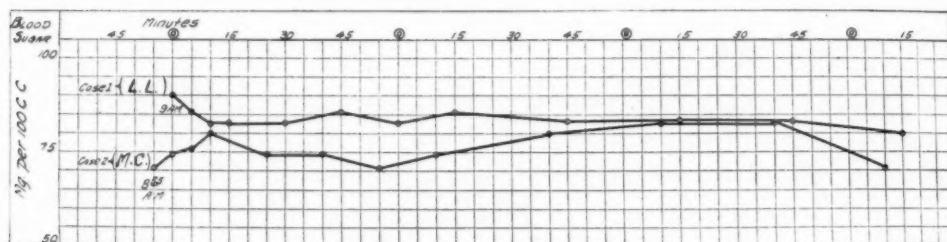

Chart I.—Blood-sugar curves in two normal, nontoxic pregnant women near term; specimens taken after overnight fast. No fluctuations and comparatively little variation in three and a half hours.

various conflicting ideas. The misleading results of taking specimens too infrequently is illustrated by the typical cases (2 and 3) plotted in Chart VII. We show these curves in order that any other investigators desiring to carry out similar studies will avoid these conflicting errors.

Following a convulsion, the patient usually has a more or less temporary rise in blood sugar, undoubtedly the effect of the enormous muscular activity of the convulsion itself. The physiologic response of the liver to any muscular activity is to throw out glycogen from its reserve stores, and blood sugar is thereby immediately increased. In eclampsia, there is a constant tendency toward remissions to lower levels so that the general trend of the sugar content of the blood was found to be downward, a phenomenon which we attribute to a steady exhaustion of the liver's glycogen reserve.

It was to be foreseen that several factors would influence or affect the level at which blood sugar is found at the beginning of the study of an eclamptic. Some of these are (1) the general state of the patient's nourishment before the attack, (2) the number of convulsions

which had already occurred, (3) the severity of her convulsions, and (4) the frequency with which they were recurring. Hypothetical variations from normal toward hypo- or hyperactivity of the patient's pancreas in its insulin-producing properties has also been considered by us as being of possible importance but cannot be discussed at this time.

The temporary increase in blood sugar from muscular exertion is augmented in eclamptics by two additional factors having the same effect, according to Stander;[19] namely, periods of moderate asphyxia plus the morphine which is customarily administered quite promptly in the routine treatment of these patients. The fact that we found a glycemia above 125 mg. in only three of our cases is additional evidence, therefore, of the correctness of our contention that hyperglycemia is not characteristic of eclampsia.

It is not surprising, however (to quote again from our preliminary report), that various investigators have been misled to believe and advance the dictum that hyperglycemia is the rule in eclampsia, whereas we interpret it as being merely an incidental and temporary effect of these factors mentioned above. The prevailing view at the present time is probably due to the fact that only one or two specimens are ordinarily taken in a more or less perfunctory way during the course of an attack of eclampsia (usually following a convulsion) and the sugar levels found in these considered as representative of the entire case, and also of the disease.

So far as we can learn no such studies of blood-sugar curves have hitherto been made either in toxemia of pregnancy or even in normal pregnant women, and we believe that this work now establishes a definite association between disturbances in metabolism and the convulsions of eclampsia. Moreover, we already have reason to believe that it opens up a field of investigation which should throw new light on the etiology of other toxemic disturbances during pregnancy, even to the extent of establishing a relationship between them all.

Blood chemistry studies which included similar serial readings of nonprotein nitrogen, urea, and uric acid were made in a sufficient number of instances to establish that in these three constituents there is no fluctuation at all comparable to that of the blood sugar.

We are now prepared to make the statement that in eclampsia it is characteristic for the convulsions to be preceded by periods of relative hypoglycemia.

EXPLANATION OF THE TERM "RELATIVE HYPOGLYCEMIA"

A blood-sugar level of 140 mg. per 100 c.c. (as in Chart II) may be considered a relative hypoglycemia when it had been 175 mg. only twenty minutes before, and 222 mg. thirty minutes before that. Likewise a fall from 152 mg. to 117 mg. in an interval of only twenty

minutes is to be characterized as a sudden relative hypoglycemia. In Chart III there occurs a fall from 89 to 54 mg. within a period of fifteen minutes, and in this instance a convulsion would seem to be the thing to expect as a natural sequence of events. It took place at about this level after a momentary recovery as indicated in Chart III.

In Chart IV there is, within twenty minutes, a drop from 82 to 46 mg., followed by a convulsion. About an hour later the reading is 75 mg. per 100 c.c. of blood, but twenty minutes after this a convulsion occurs during which the blood sugar increases to 89 mg. This reaction is not, as it might seem, at variance with our previous deductions, because the patient had been twitchy (muscular activity) during
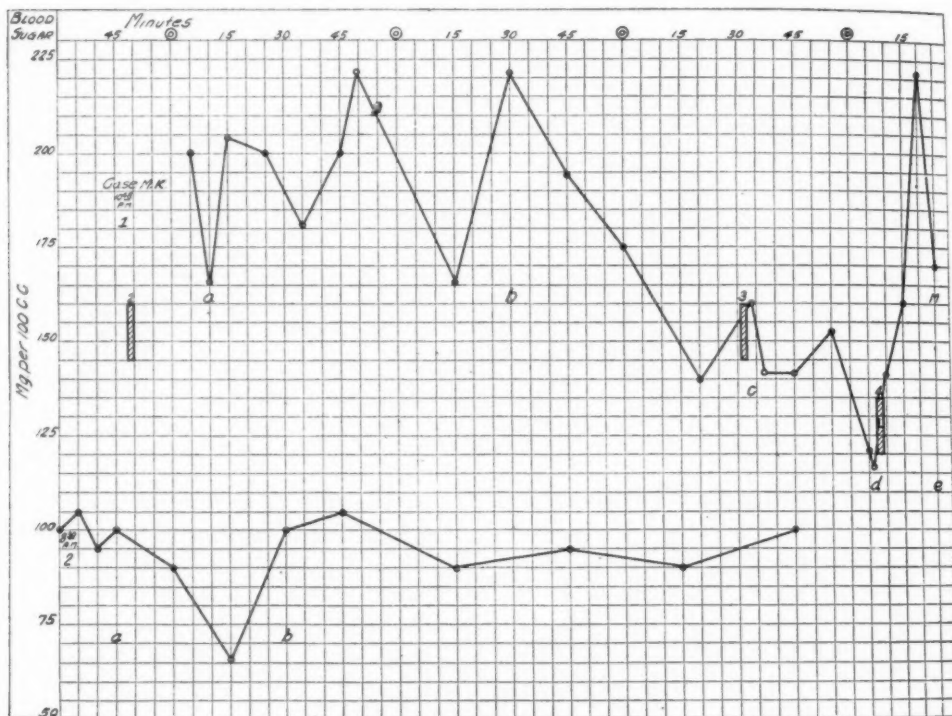

Chart II.—(1) Blood-sugar curve in patient with eclampsia. Shaded vertical bars indicate convulsions, numeral being the consecutive number, and L being the last convulsion of eclamptic attack. M indicates time medication (morphine or intravenous glucose or both) was begun.

Initial level 200 mg., highest level 222 mg., lowest level 117 mg. sugar per 100 c.c. of blood.

Period a to b: Interval of marked fluctuations but no convulsions.

Period b to c: Development of sudden relative hypoglycemia followed by convulsion.

Period c to d: Temporary hyperglycemia resulting from convulsions, followed by further hypoglycemic remission and convulsion.

Period d to e: Sharp recovery to hyperglycemic levels, followed by equally sharp fall. Beginning of medication at this point presumably prevented another imminent convulsion.

(2) Blood-sugar curve on same patient three weeks later. Not entirely free from albuminuria; blood pressure occasionally fluctuating upward but averaging 128/90. Patient still shows after-effects of eclamptic seizure but has practically recovered. Blood specimens collected after overnight fast, with exception of one downward fluctuation (a to b), are practically normal.

this interval. Moreover, five minute readings were not being taken, so that further information which might change the picture was not available. Even in Case 2, Chart VI, where convulsions were occurring so frequently as to be confusing, unless readings could be made at intervals of a minute or two, fall after fall preceded them.

It is desired at this point to call attention again to the sharply contrasting curves taken for control in normal pregnant women near term.

Based on general experience with insulin overdosage, both experimental and clinical, the tendency is to think of hypoglycemic symp-
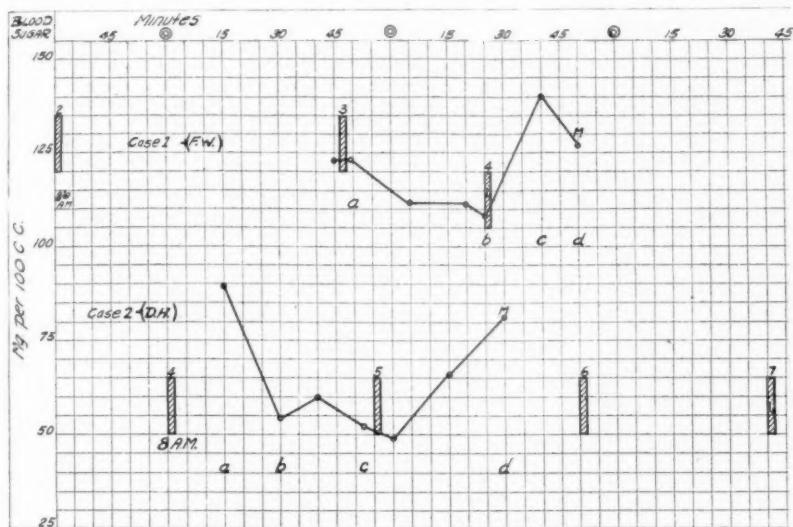

Chart III.—Same symbols as in Chart II.

Case 1: Blood-sugar curve in active eclampsia.
Initial level 123 mg., highest level 140 mg., lowest level 108 mg. sugar per 100 c.c. of blood.
Period *a* to *b*: Moderate drop in blood sugar (15 mg. in thirty-five minutes) followed by convulsion.
Period *b* to *c*: Relative hyperglycemia as reaction from convulsion.
Period *c* to *d*: Probable beginning of remission; medication begun.
Case 2: Blood-sugar curve in active eclampsia.
Initial level 89 mg., highest level 89 mg., lowest level 48 mg. sugar per 100 c.c. of blood.
Period *a* to *b*: Hypoglycemic fall from 89 to 54 mg. in fifteen minutes.
Period *b* to *c*: Convulsion imminent at point *b*, avoided by momentary reaction, was followed by further fall and convulsion.
Period *c* to *d*: Hyperglycemic reaction from convulsion; medication begun.
Two additional convulsions without blood-sugar data.

toms (nervousness, tremor, twitching, and convulsions) as occurring only at or below the level of 70 mg. of sugar per 100 c.c. of blood, or thereabouts.

MacLeod[20] makes an interesting observation in this connection, however, which has a bearing on our studies. He says in effect that the level at which hypoglycemic symptoms follow insulin administration may depend not so much on any absolute level of blood sugar as it does on the rapidity with which that level is reached. In other words,

blood sugar might even be reduced to 45 mg. or less without convulsions if that level is slowly attained, whereas convulsions can easily be produced at 75 or 80 mg. or even higher levels, if this point is produced quickly enough by sudden large, or frequently repeated smaller doses of insulin.*
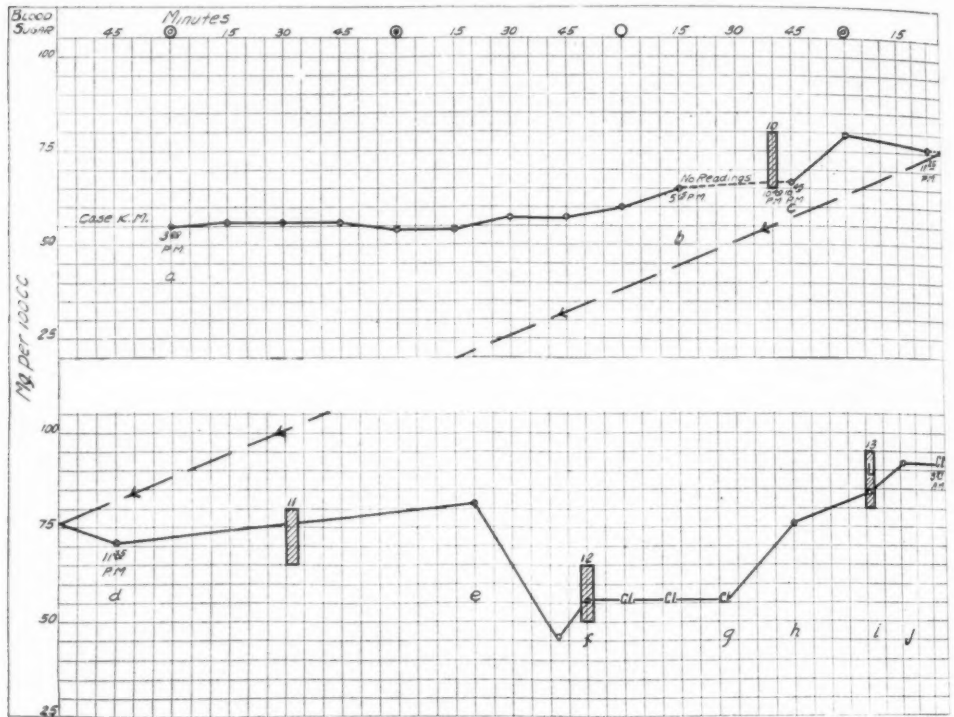

Chart IV.—Blood-sugar curve in slowly progressing case of eclampsia. Low blood sugar on admission, following nine convulsions (presumably glycogen depletion); patient quiet from morphine administered at home.

Initial level 55 mg., highest level 92 mg., lowest level 48 mg. sugar per 100 c.c. of blood.

Period *a* to *b*: No fluctuation, no convulsions for two and a quarter hours; morphine still effective; patient apparently recovering.

Period *b* to *c*: Interval of five and a half hours. Specimens discontinued because no symptoms until convulsion at 10:40 P.M.

Period *c* to *d*: (Arrow-marks continue graph to lower panel of chart.) Temporary increase followed by slow drop in blood sugar. Fluctuations not marked; convulsions infrequent.

Period *d* to *e*: One and a half hours with no blood specimens, but one convulsion.

Period *e* to *f*: Hypoglycemic fall (82 to 46 mg. in twenty-two minutes), followed by convulsion.

Period *f* to *g*: Specimens clotted.

Period *f* to *i*: No data on glycemia curve except single reading at point *h*. Might have shown expected peak preceding point *i* if figures were available.

Period *i* to *j*: Only information gained because of incomplete data between *f* and *j* is that convulsion 13 (*L*) was followed by slight rise in blood sugar.

Summary: Case demonstrates relation between degree and frequency of fluctuations and occurrence of convulsions, both being comparatively inactive; also that convulsions 10 and 13 were followed by rise in blood sugar, whereas the one convulsion (12), before which suitable specimens were obtained was preceded by a sharp fall.

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*A paper by John (Am. Jour. Med. Sc., 1926, clxxii, 96), not seen by us until recently, reports 24 cases of toxic insulin reactions in diabetics at normal or higher than normal blood-sugar levels. In five of these patients the blood sugar was over 200 mg. per 100 c.c. when the reactions occurred. Remembering that they must have been higher when the insulin was given, it is reasonable to say that they might be classed under our term "relative hypoglycemia."

GENERAL DEDUCTIONS

As a result of our clinical and experimental studies[3, 6, 21] of those toxemic disturbances seen in the early, as well as those in the late weeks of pregnancy, we are convinced (1) that there is a definite relationship between the two, (2) that the difference between the hepatic lesions of the two states is less distinctive than has been generally supposed, (3) that there is no specific toxin of fetal origin responsible for these toxic states, (4) that eclampsia in particular and other toxicoses of pregnancy as well are due entirely to disturbance in maternal metabolism, (5) that nitrogenous metabolism plays no rôle in this,
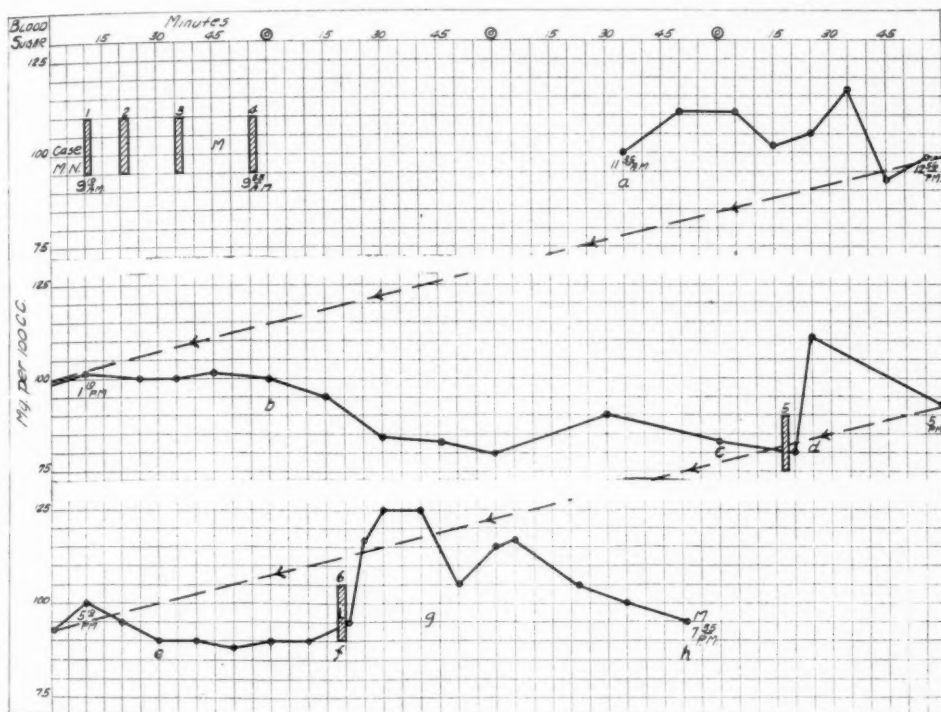

Chart V.—Blood-sugar curve in slowly progressing case of eclampsia.

Initial level 100 mg., highest level 125 mg., lowest level 80 mg. sugar per 100 c.c. of blood.

Between third and fourth convulsion patient had been given morphine, gr. ½, by hypodermic, and chloral hydrate, gr. xxx, by rectum.

Period *a* to *b*: (Arrow-marks continue graph to middle panel.) No marked fluctuations; patient resting quietly; narcotics apparently still effective.

Period *b* to *c*: Gradual decline in blood sugar. Clinically patient seems improved, arousing occasionally and asking questions. At point *c* no clinical warning of impending convulsion but blood sugar proved to be at low ebb.

Period *c* to *d*: Convulsion caused sharp rise in blood sugar.

Period *d* to *e*: (Arrow-marks continue graph to lower panel.) Remission downward more rapid than in period *b* to *c*.

Period *e* to *f*: Low level of blood sugar might have caused convulsion 6 (*L*) to occur at any point in this period.

Period *f* to *g*: Sharp rise following convulsion.

Period *g* to *h*: Rapid remission downward; convulsion probably imminent.

Point *h*: Medication begun; morphine, gr. ¼, by hypodermic, and intravenous injection of glucose (200 c.c. 25 per cent solution in one hour, ten minutes). No more convulsions.

and (6) that this disturbance is one of carbohydrate metabolism, based primarily on a deficiency in carbohydrate intake plus increased consumption of carbohydrates resulting in a depletion of the glycogen stores with consequential damage to the liver and its functions.

In corroboration of these assertions we wish to assemble and correlate as briefly as possible a small part of the immense amount of investigation which has been done on this and related subjects.

Regarding the first point, both hyperemesis and eclampsia (to consider only the outstanding "toxemias" of early and of late pregnancy) have pregnancy in common. The most distinctive pathologic change in fatal cases of each of them are focal necroses of the liver lobules.
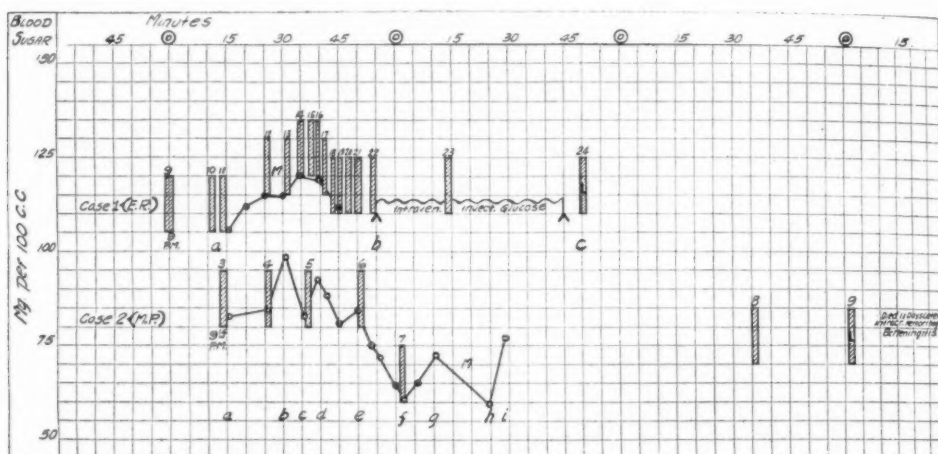

Chart VI.—Blood-sugar curves in two rapidly progressing cases of eclampsia, with frequently recurring convulsions.

Case 1: Convulsions recurring at such frequent intervals (60 to 90 seconds apart) that comprehensive data were not obtainable.

Initial level 106 mg., highest level 120 mg., lowest level 106 mg. sugar per 100 c.c. of blood.

Period b to c: Illustrates effect on convulsions of intravenous injection of hypertonic (25 per cent) glucose solution.

Case 2: Fluctuations present but their degree affected by frequency of convulsions.

Initial level 83 mg., highest level 98 mg., lowest level 59 mg. sugar per 100 c.c. of blood.

Period a to b: Two convulsions in eleven minutes, followed by rise in blood sugar.
Period b to c: Fall in blood sugar; convulsion.
Period c to d: Rise following convulsion.
Period d to e: Fall in blood sugar; convulsion.
Period e to f: Sharp fall (84 mg. to 65 mg.) in ten minutes.
Period f to g, g to h, h to i: Further fluctuations without convulsions; medication begun.

We now have work nearly ready for publication which demonstrates a marked carbohydrate depletion or deficiency with hypoglycemia the outstanding feature of the blood picture in hyperemesis, while this present report deals with the fluctuations in blood sugar during eclampsia and the relation of the hypoglycemic levels to the actual production of the convulsions. Therapeutically the intravenous administration of hypertonic glucose solution has been proved to have

immediately beneficial effect in both conditions. Many authorities, notably Freund,[22] believe in such a relationship, and it is not unreasonable.

Regarding, secondly, the pathologic changes noted, it has been said that there is a distinctive histologic difference between the two conditions. Our own work[6] not only failed to demonstrate such a contrast but also showed that the usual pathologic picture was altered in women who had received glucose injections. Bell[23] concludes after a careful histologic study of toxemia of pregnancy in which the out-
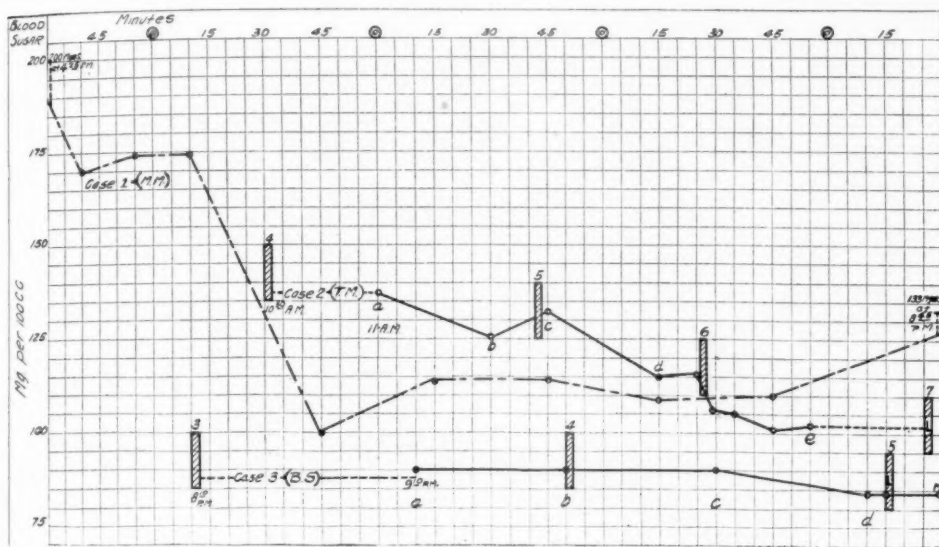

Chart VII.—Three of our earlier cases; inconclusive because technic not yet properly developed and specimens taken too infrequently.

These cases are shown to illustrate what seemed at first to be results contradictory and discouraging to our theories, but which later proved to be due to faulty technic in our study of the cases.

Case 1: Initial level 200 mg., highest level 200 mg., lowest level 100 mg., sugar per 100 c.c. of blood.

Two convulsions occurred, but by an unfortunate error the exact time at which they took place was recorded only on the nurses' record. In filing the history, the nurses' record was destroyed, so that this graph permits no conclusions. It shows, however, an interesting fluctuation which is similar to those of the other more complete cases.

Case 2: Inconclusive case because of infrequent readings at critical times.

Initial level 137 mg., highest level 137 mg., lowest level 102 mg. sugar per 100 c.c. of blood.

Period *a* to *b*: Moderate fall; convulsion twelve minutes after *b*.

Period *b* to *c*: Point *c* slightly higher than *b*.

Period *c* to *d*: No readings to show probable rise and fall but *d* is 10 mg. lower than *b* and 17 mg. lower than *c*. This period represents an unfortunate omission.

Period *d* to *e*: Convulsions without blood-sugar increase in response. This is apparently contradictory but is to be explained as in other instances as due to advancing depletion of glycogen reserve stores.

Period *e* to last convulsion: No specimens taken.

Case 3: Entirely inconclusive because specimens *a* and *b* were taken just as convulsions began, with no intervening specimens.

Initial level 90 mg., highest level 90 mg., lowest level 89 mg. sugar per 100 c.c. of blood.

Specimen at *c* taken forty minutes after convulsion instead of five minutes later as should have been done.

Specimen at *d*, after another forty minute interval, but five minutes before a convulsion.

Period *c* to *d* would probably have shown some interesting fluctuations had specimens been taken more frequently during this time.

standing features which he found were fatty infiltration, infarction, and hemorrhagic necrosis that his "data weakens our belief in any one lesion of the liver being considered essential for toxemia of pregnancy." Finally, similar liver lesions occur in fatal cases of simple starvation.

The third conclusion, against there being a specific fetal toxin, arises from the fact that in spite of a vast amount of work designed to
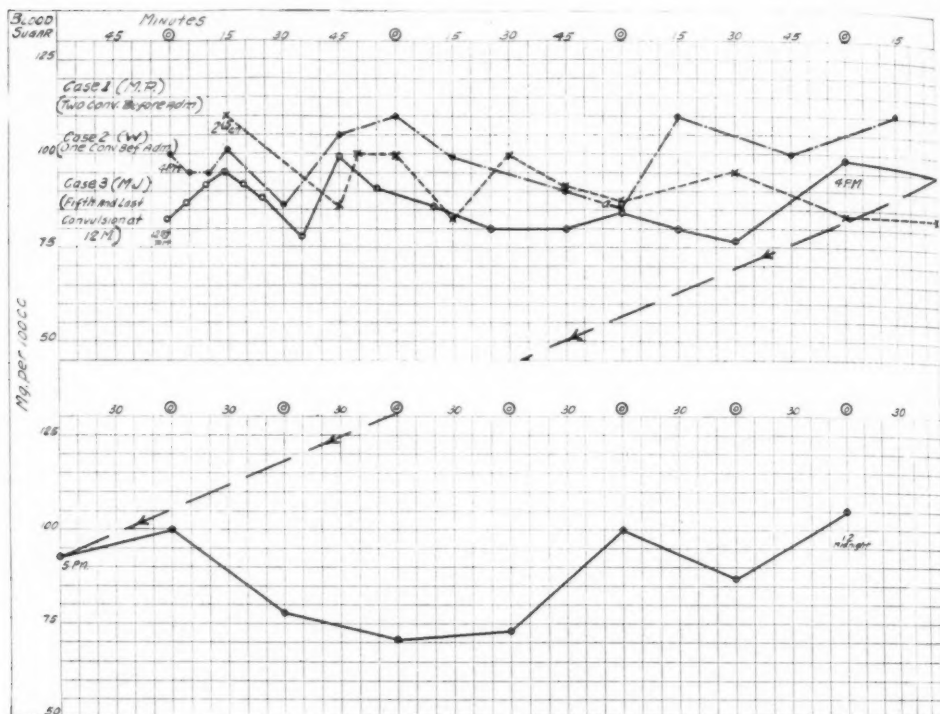

Chart VIII.—Cases illustrating persistence of fluctuations after convulsions have ceased.

Case 1: (Dotted line) Two convulsions before admission; none after admission.
Initial level 110 mg., highest level 110 mg., lowest level 83 mg. sugar per 100 c.c. of blood.
Downward fluctuations fairly sharp but followed by quick recovery to higher and safer levels, without convulsions. Curve more nearly level and steadier toward end of study.

Case 2: (Dot and dash line) One convulsion before admission, none after admission.
Initial level 100 mg., highest level 111 mg., lowest level 87 mg. sugar per 100 c.c. of blood.
At point a, patient became twitchy, and a convulsion seemed imminent. Blood-sugar readings showed that this irritability was preceded by a fall in blood sugar, and was followed by a sharp rise. These results indicate that a convulsion had been narrowly averted.

Case 3: (Solid line) Fifth and last convulsion took place at 12 M., shortly after admission; blood-sugar series was begun one hour later and continued until midnight.
Initial level 83 mg., highest level 105 mg., lowest level 71 mg. sugar per 100 c.c. of blood.
In upper panel of chart each vertical line represents five minutes, in lower panel ten minutes. Specimens were taken more infrequently as time passed, and it was apparent that the patient's condition was improving.
No medication was given this patient during the time of this study, and it is obvious that she, like all eclamptic patients, was necessarily in a fasting state, since coma is an almost constant characteristic of the disease.

develop and prove such an idea, there is still no evidence to show that the fetus produces any toxin capable of causing eclampsia. Moreover, the occurrence of eclampsia with hydatidiform mole has been reported many times and not only serves to eliminate the fetus from responsibility but also to strengthen the carbohydrate deficiency theory. Such a mole is an enormous overgrowth of chorionic or placental tissue. Analysis of fetal tissues shows that the placenta exceeds all other fetal organs in its glycogen content[24] and to produce such a mass of chorionic tissue as represented by a mole must argue a glycogen deprivation elsewhere.

Dieckmann and Crossen[25] in their recent comprehensive work comment on the glycogen requirements incidental to the extremely rapid multiplication in the size of the fetus as pregnancy progresses as well as that due to the tremendous hypertrophy of the uterus. They conclude most logically "that systematic study of the metabolism particularly of the carbohydrates, * * * will enlighten us more as to the etiology, pathology, and treatment than speculations concerning 'toxins' or deranged glandular function."

The occurrence of postpartum eclampsia would seem to eliminate both fetus and placenta from direct responsibility and may be explained as being an aftermath of their inordinate demands.

The fourth and fifth points are generally conceded and have been discussed elsewhere in this paper, while the sixth is the theme of this essay.

AUTHORITATIVE RELATED OPINIONS AND RESEARCH

Even at the risk of unduly lengthening this paper, it is necessary here to refer to and accredit the work of those investigators whose research has a bearing on these present studies.

Benthin[26] refers to Hofbauer's work on glycogen depletion of the liver cells, and says that "through these changes in liver tissue a disturbance in function develops . . . . bearing on carbohydrate metabolism." He also says "the viewpoint that when disturbances in metabolism are observed, they must stand in some relation to the liver is as unassailable as the following: that with physiologic and pathologic changes in the liver tissue metabolic disturbances make themselves noticeable." He likewise noted that blood-sugar values in eclampsia showed an increase only when the blood was taken after a convulsion or between two convulsions, but he did not demonstrate the fall preceding the convulsion which we consider the essential motivating factor. In summarizing his beliefs he holds that a damaging disturbance of liver function is etiologically involved in the origin of eclampsia (Eine Funktionsschädigung der Leber ätiologisch für die Genese der Eklampsie in Betracht zu ziehen).

Opitz[27] notes the similarity between certain features of pregnancy toxemias and "hunger-diseases"; he speaks also of the similarity of the reactions in pregnancy to those observed in patients with large malignant growths and severe infections (increased antitrypsin in the blood, more rapid sedimentation of blood corpuscles, similarity in course of Abderhalden ferment reactions, alterations of cobra-venom hemolysis, the complement fixation, and the diminution of the colloidal protective action of the serum). He concludes that toxemia may be due to the following

combination: pregnancy to be compared to a rapid-growing malignant tumor, plus hunger through decreased intake of important nutritive elements, and that pregnancy toxemia is the result of disturbed metabolism resembling the various hunger-diseases more than any other group of conditions.

The relation between the glycogen content or depletion of the liver and the histologic changes has been referred to: Dudley and Marrian[18] found not only that insulin given to normal animals failed to accelerate the glycogen storage in the liver but rather that the livers of normal animals which had received sufficient insulin to bring them to the verge of hypoglycemic convulsions contain very little glycogen. Glycogen depletion is followed by fatty infiltration and disturbance of liver function. Barbour, Chaikoff, MacLeod, and Orr[28] of the Toronto group, who at first thought otherwise, now subscribe to this view.

Likewise, Mann and Magath[29] have repeatedly demonstrated that the most marked result of experimental extirpation of an animal's liver is a progressive fall in blood sugar. With its continued fall the expected symptoms of hypoglycemia develop as in "extirpation" of the liver glycogen by insulin, convulsions follow, and in one of them the animal dies. If, however, during any stage after development to the point at which respiration has actually stopped, glucose is injected, the animal immediately and completely recovers. This work appeared some time after our original theories and recommendations regarding the use of glucose injections in hyperemesis and eclampsia were first published, but has a remarkable relation to all that we then and now have to say.

Obata and Hayashi[15] in some experimental work with strychnine poisoning in animals demonstrated rises in blood sugar after the convulsions with remissions between them. They commented that similar conditions might prevail in eclampsia but failed to carry out to a conclusion such studies in eclamptic women.

Correlation of the foregoing with our own work makes it possible to deduce that: The needs of the fetus create a glycogen demand which with other factors previously referred to, accounts for the removal of glycogen from the liver; the histologic structure of the latter is changed in proportion to its depletion; this destruction of liver tissue becomes equivalent presently to its partial "extirpation"; carbohydrate metabolism is thereby affected; the blood-sugar values begin to seek hypoglycemic levels followed by frenzied efforts toward recovery thus initiating the fluctuating waves which have been noted in the curves of our charts; convulsions follow at certain low levels as the fluctuations become more and more violent, but like the familiar hypoglycemic convulsions these too can be controlled by glucose injections.

The usual nephritis of preeclampsia and eclampsia may be considered more or less of an incident, as it is in any profound disturbance of metabolism, or intoxication resulting from either a bacterial or a chemical poison. Similarly it will be remembered that nephritis occurs as the result of severe hyperemesis gravidarum, while in its more fulminating state, known as acute yellow atrophy of the liver, where the liver destruction is rapid and extensive, typical "eclamptic" convulsions often occur—still further evidence of relationship between these states. The underlying metabolic disturbances are strikingly alike. Preexisting chronic nephritis probably increases the patient's susceptibility to the development of "toxemia" during pregnancy, thus being a predisposing factor.

LABORATORY PROCEDURES

Due precautions have been taken in the laboratory work to eliminate all possible sources of error. Obviously all blood specimens were taken on a "fasting" basis, since no woman in eclamptic coma is taking nourishment, and our normal controls were fasted overnight.

In certain of our earlier cases calcium oxalate was used as the anticoagulant, and in these series if the examinations could not be made at once, the filtrate was prepared immediately and only then held for later examination. Since that time we have invariably used sodium fluoride (10 parts) and thymol (1 part), which combination prevents deterioration of blood-sugar values. As the serial readings were made, new standards were prepared for every six specimens which were done at one time.

EFFECT OF THESE STUDIES ON THE USE OF INSULIN IN PREGNANCY TOXEMIAS

Endogenous insulin production is supposed to fluctuate in response to food intake or to abstinence from food.

It has been shown[30] and confirmed[31, 32] that insulin production in a normal individual is stimulated by intravenous injections of glucose solution. Thalhimer and others[33] have also demonstrated that this can be carried to such an extreme of overstimulation as to produce the following apparently paradoxical situation: the intravenous injection of a continuous stream of glucose solution at a slow rate over a certain period of time causes blood sugar first to rise, then steadily to fall as the pancreas responds to the injection by increased insulin production, this fall continuing to such a low level in spite of the constant inflow of glucose that presently symptoms of hypoglycemia manifest themselves and blood-sugar readings correspond to those seen in insulin overdosage. We would comment that for this hypoglycemia there is no antidote, and these findings emphasize our earlier contention that for therapeutic purposes single large injections of glucose solution repeated at stipulated intervals are preferable to continuous injections over any considerable period of time.

In a woman with toxemia of pregnancy whose insulin producing ability is supposedly unimpaired even though temporarily in abeyance as the result of a carbohydrate deficiency, the mere injection of glucose is sufficient therefore to stimulate her pancreas in this respect. If it be conceded that her insulin production is temporarily restricted, it must be remembered that this is a response in a normal physiologic way to the temporary deficiency in carbohydrates and thus is a protective measure.

If Nature responds to the glucose injection with an overdose of endogenous insulin, every unit injected by a hypodermic syringe becomes a still greater overdose if the woman is, as we suppose, not a diabetic.

The rationale of adding insulin to intravenous injections of glucose for nondiabetic intoxications and acidoses is not consistent, and now that we have demonstrated a disturbance in carbohydrate metabolism with hypoglycemia (the opposite of diabetes) it is obvious that this procedure is contraindicated.

Those cases in which benefit has been reported from the combination of glucose and insulin were possibly accelerated momentarily by the extra insulin, but the benefit was derived from the excess of glucose (overprotection) rather than from the injected insulin. Glucose alone would have been safer, while with the pancreas normal as it undoubtedly was, the injected insulin was unnecessary. Each succeeding glucose injection stimulates the endogenous insulin production to further and further activity, so that the addition of insulin to subsequent injections becomes increasingly dangerous.

The suggestion of Stander and Duncan previously referred to; namely, that insulin alone or with merely a ''protective dose of glucose'' should be given to eclamptics even in the absence of bloodsugar readings was undoubtedly the outgrowth of Thalhimer's[34] addition of insulin to the glucose therapy of the disease.

This use of insulin in pregnancy toxemias has been vigorously opposed by Titus,[35] by Harding,[36] and recently by Bokelmann,[37] of Berlin. MacLeod and Campbell[38] also disapprove of insulin being added to glucose in the treatment of acidosis resulting from this and all other nondiabetic states.

The interesting thought had occurred to us in the course of this study that an overproduction of maternal insulin from some unknown cause might have a bearing on the etiology of hyperemesis, since in these cases there is usually a hypoglycemia (to be published in later report), and that the development of the fetal pancreas with its own insulin production might play a rôle in the etiology of eclampsia. These ideas have been discarded, however, for the more reasonable ''carbohydrate deficiency theory'' as originally outlined. There is no evidence to warrant the idea that the maternal pancreas becomes overactive in early pregnancy, and in hydatidiform moles with eclampsia there is no fetal pancreas.

CONCLUSIONS REGARDING TREATMENT OF PREECLAMPSIA AND ECLAMPSIA

For preeclampsia the usual rational procedures (restriction of salt and of protein intake, rest, attention to bowels and kidneys, etc.) are to be vigorously utilized, and in view of the disturbance in carbohydrate metabolism now demonstrated, increased carbohydrate intake should immediately be instituted. These patients do not need intravenous injections of glucose, because they are able to take and retain food by mouth. A diet high in carbohydrates is essential for preeclampsia, therefore, and glucose solution with fruit juices should be one of its features.

The so-called conservative treatment of eclampsia, advocated by Stroganoff, by Tweedy of the Rotunda school, and by Williams, has by its lowered mortality rates thoroughly proved its value over the practice of undertaking active interference and immediate operative delivery of the fetus during eclampsia.

As a result of our studies we believe that a reasonable basis is now established for what have been empirically successful methods. With these findings in mind we wish to outline the therapeutic procedures which now seem essential in the treatment of eclampsia:

First, the administration of morphine by hypodermic injection, or of magnesium sulphate (Lazard[39]) by hypodermic or intravenous injection, and of chloral hydrate by rectum. Each of these drugs is of value in eclampsia, although they differ somewhat in their general effect. Their sedative action checks the convulsions and by producing absolute muscular rest and relaxation, the patient is afforded an opportunity to restore her metabolic equilibrium.

The action of morphine is prompt, but the effect is less lasting than magnesium sulphate. Chloral has an advantage over morphine for continued use during the later hours of the first day or two of an attack (after convulsions have subsided) in that its by-effects are less obnoxious. Magnesium sulphate (intravenously) has an effect in addition to its sedative action, according to Lazard, of reducing cerebral edema and of stimulating elimination.

Second, the intravenous administration of hypertonic glucose solution in single doses of 75 gm. in 300 c.c. of water (25 per cent solution) at the rate of not more than 1 gm. per minute, or from an hour and a quarter to an hour and a half for the entire injection. This should be repeated after intervals of four or five hours during the attack and for a time following cessation of the convulsions (three or four times in first twenty-four hours, according to the needs and response of the patient) until fluids can be taken freely by mouth.

The symptomatic treatment of the eclamptic convulsions having been accomplished by the sedatives, the use of glucose is directed toward the underlying cause of the disease. It is antidotal to the hypoglycemia; it has a liver-sparing effect, restoring glycogen depletion of the liver, and it furnishes both glucose and water to the tissues.

The immediately noticeable effects are diuresis, lowering of blood pressure directly following the injections, cessation of the convulsions, and the prompt regaining of consciousness. The odor of acetone quickly disappears from the breath, and edema lessens rapidly after the injections.

Interference with pregnancy is to be undertaken only according to the present tenets of the "conservative" treatment. If labor supervenes, the patient is to be delivered, preferably by forceps in the

second stage (nitrous oxide and oxygen, or ether anesthesia) with as little disturbance to her as possible. If labor does not begin during the eclampsia, all attempts at operative interruption of the pregnancy are interdicted until at least hours and preferably days have elapsed following the cessation of the convulsions. In a certain percentage of instances pregnancy may be allowed to continue, in others the patient's condition makes it necessary eventually to interfere by induction of labor.

A curious fact which we have observed during the past few years of work on this subject is the decreasing frequency of eclampsia. Prenatal care seems to be steadily diminishing its incidence, although this alone cannot be expected entirely to stamp it out, but in the larger centers it is being seen more and more infrequently. This opinion represents a fairly general survey because in Pittsburgh many of our obstetrician friends agreed to cooperate in this work and to call us to their hospitals whenever a case appeared. However, the cases were so limited in number that two of us (Titus and Dodds) on different occasions made this the excuse for fairly protracted stays in New York and Chicago where we supposed the very size of the cities would make for a plentiful number of cases. Even there we found the same general condition prevailing.

It is a splendid commentary on the spirit of cooperation in our profession toward work of this sort that in these two large cities our many friends without a single objection agreed and arranged to make available to us for study any cases they had, without any more than the mere assurance that this was a carefully planned investigation.*

It is also satisfactory to relate that with but one exception every patient on whom these studies were made recovered from her eclampsia. This one patient lived for twelve days after recovering from the eclamptic seizure and then died with symptoms of intracranial hemorrhage and meningitis. It was apparent, therefore, that no undue risks were taken with these patients by carrying out this study.

No conclusions as to mortality rates should be drawn from any but large series of cases, but with these present methods of treatment the admission of a patient with eclampsia to our hospital service is now viewed with considerable equanimity rather than with the alarm which such a condition formerly aroused.

We desire to express our appreciation to those who have been of assistance to us in the laboratory work connected with this study: Mr. Nerses, Dr. Edmund Smith, and Dr. Wiener of New York, Dr. Sarraff, Dr. Tefft, Miss Fichtel, and Miss Egbert, of Pittsburgh.

CONCLUSIONS

1. Evidence is now offered to demonstrate that in eclampsia the disturbance in carbohydrate metabolism, heretofore assumed, actually exists.

2. This disturbed metabolism is apparently the result of the carbohydrate deficiency in pregnancy which we have previously postulated.

3. Contrary to the general opinion, hyperglycemia is not characteristic of eclampsia.

4. Serial blood-sugar readings during an attack of eclampsia show wide fluctuations in exceedingly short intervals of time. Similar series in normal pregnant women near term show no such variations. Graphs of such glycemia curves are shown to substantiate the foregoing statement.

5. The convulsive seizures occur at levels which we have designated as "relative hypoglycemia," and apparently are caused by the sudden drops in blood sugar now being reported. In this respect eclamptic convulsions are comparable to those of insulin hypoglycemia.

6. Following a convulsion, there is usually a temporary rise in blood sugar, the customary physiologic response of the liver to muscular activity.

7. There exists a tendency toward remissions to lower levels so that the general trend of the sugar content of the blood in eclampsia is downward, obviously the effect of exhaustion of reserve glycogen stores in the liver.

8. As a result of this and previous studies of toxemic disturbances of pregnancy, we are led to conclude (1) that there is a relationship between all toxemias of pregnancy, (2) that the difference between the hepatic lesions of the various clinical states is less distinctive than has been generally supposed, (3) that there is no specific toxin of fetal origin responsible for these toxic states, (4) that the various toxicoses of pregnancy, particularly eclampsia, are due entirely to disturbance in maternal metabolism, and (5) that this disturbance is one of carbohydrate metabolism, based primarily on a deficiency in carbohydrate intake plus increased consumption of carbohydrates which results in a depletion of the glycogen stores with consequential damage to the liver and its functions.

9. This glycogen deficiency in the liver presently becomes equivalent to its partial "extirpation"; the blood-sugar values begin to seek hypoglycemic levels followed by frenzied efforts toward recovery, thus initiating the fluctuating waves noted in our charts; the convulsions which occur at certain low levels as the fluctuations become more and more violent are controllable, like the familiar hypoglycemic convulsions, by glucose injections.

10. The nephritis of preeclampsia and eclampsia, as well as of grave hyperemesis, is not the forerunner but an incidental symptom and result of the intoxication.

11. The insulin production of a nondiabetic pancreas may be temporarily in abeyance during a pregnancy intoxication as a physiologic response to the lessened glycogen reserve in the body. Such a pancreas should respond to an injection of glucose as does any normal pancreas, by an overproduction of endogenous insulin so that any additional insulin injected is an additional overdose.

12. With the view that the convulsions of eclampsia are to be designated as a hypoglycemic reaction or manifestation, the use of insulin either with or without glucose in the treatment of this disease is unnecessary and contraindicated.

13. Appropriate treatment for eclampsia as established by these glycemia-curve studies consists of two main features (a) complete muscular rest and relaxation as induced by quiet, isolation, and the use of morphine by hypodermic injection, chloral hydrate by bowel, and magnesium sulphate by hypodermic or intravenous injection, and (b) the intravenous injection of strongly hypertonic glucose solution. It is urged that the prevalent underdosage be avoided and sufficient glucose in sufficient concentration be given to produce a full therapeutic effect.

14. The statement made in earlier communications is reiterated here that the therapeutic dose of glucose for an adult is a minimum of 50 to 75 gm. in 200 to 300 c.c. of distilled water respectively (25 per cent solution) given by intravenous injection over a period of not less than one hour and repeated after intervals of from four to five hours according to the needs and response of the patient.

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1015 HIGHLAND BUILDING.

THE PLACE OF THE VAGINAL CESAREAN SECTION IN OBSTETRICS

BY LOUIS E. PHANEUF, M.D., F.A.C.S., BOSTON, MASS.

DÜHRSSEN, in 1890, recommended the use of deep incisions in the anterior or posterior lips of the cervix, or both, but avoiding the lateral portions, in order that the deep, irregular cervical lacerations which are likely to follow a rapid delivery through a rigid undilated cervix might be avoided. It was soon discovered, however, that the incisions did not reach high enough to overcome the resistance of the internal os, that extensive tears which were difficult to suture were likely to follow, and that hemorrhages which were hard to control sometimes occurred.

Realizing that his incisions in the cervix did not enlarge the canal sufficiently for immediate delivery, Dührssen devised the operation which, in 1895, he described as an anterior vaginal hysterotomy, and in 1896 called "vaginal cesarean section." The original operation called for only the anterior incision; later, he modified the technic by adding the posterior incision. The result of this was that it enabled him to make a shorter anterior incision, thereby lessening the danger of injury to the bladder during delivery.

Before attempting vaginal cesarean section, certain requirements are essential. The tissues must not be edematous or friable. The uterus must be movable so that the cervix may be readily brought down into the vagina. The pelvis must be ample. The child must not be too large. In the absence of these requisites, the vaginal cesarean section should not be considered, regardless of the indication.

INDICATIONS AND CONTRAINDICATIONS

The literature of the last ten years contains very little about vaginal cesarean section. The great advantage of this method is its extra-peritoneal approach of the uterus; therefore, the marked increase in safety over the classical operation. Since the more general adoption of the suprasymphyseal or transperitoneal low abdominal cesarean
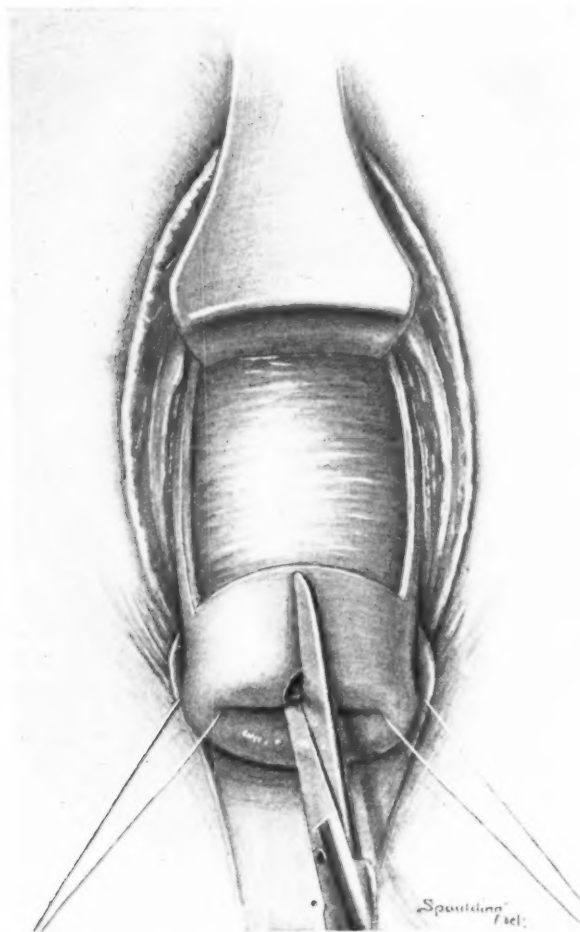
Fig. 1.—Vaginal cesarean section. The cervix is brought down by volsella. A traction suture of heavy chromic catgut is placed at each lateral angle. A transverse incision, in the anterior vaginal wall, is made at the point of attachment of the bladder to the cervix, and the bladder, together with the vaginal wall, is dissected upwards to the reflection of the anterior peritoneal culdesac. The bladder is held under a retractor, and the cervix is incised in the median line.

section, it has been found that the increased protection against infection secured by this procedure is almost as great as that of vaginal hysterotomy. For this reason, and also because of the simpler technic,

the low abdominal operation has been substituted for the vaginal in a number of clinics. This has tended further to limit the indications of vaginal hysterotomy.

Dührssen advised his operation for the following conditions:

1. *In the anomalies of the cervix and of the lower uterine segment creating some difficulties which place the mother in danger,* including cancer, myoma, ovarian tumors, and stenosis of the cervix.
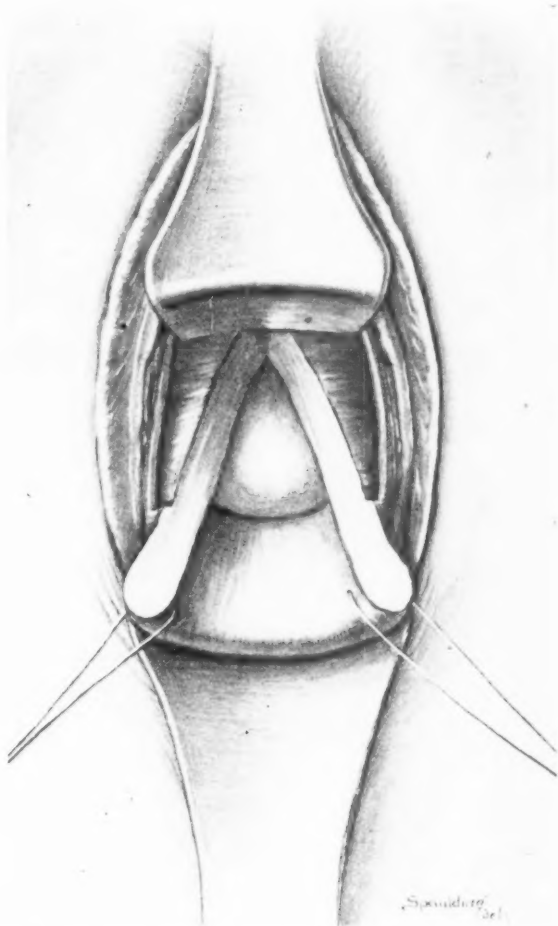
Fig. 2.—The cervix has been incised anteriorly. The membranes are shown through the incision.

2. *When the mother is in danger and when the rapid evacuation of the uterus is indicated to improve the general condition.* Lesions of the heart, lungs, kidneys; eclampsia; premature separation of the normally inserted placenta.

3. *When the mother is dead or dying.*

4. *When only the infant is in danger,* as in a slow labor and in compression of the cord.

At the present time, while vaginal cesarean section is still recognized as a valuable procedure, most obstetricians would not subscribe to the original indications, and its range of usefulness has become much more limited.

Group I.—Stenosis of the cervix remains an accepted indication. A labor complicated by myomas and ovarian tumors, however, is best terminated by an abdominal delivery followed by the ablation of the
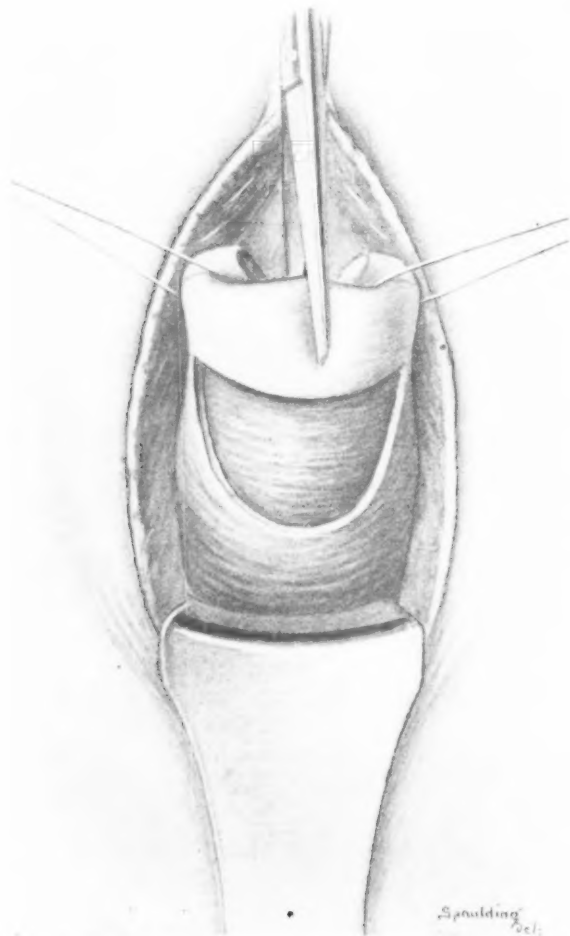
Fig. 3.—The posterior lip of the cervix is pulled upward toward the symphysis. A transverse incision is made at the junction of the vagina and cervix. The posterior culdesac and the rectum are reflected downward, and the posterior lip is incised as far up as possible, care being taken to respect the abdominal cavity.

tumor in question, or even hysterectomy in the case of multiple myomas. In cervical carcinoma, if the extension of the lesion is limited, and the patient is in good physical condition, delivery should be accomplished by the abdominal route and followed by the total extirpation of the uterus and its adnexa. In the more advanced cases de-

livery by a fundal cesarean section followed by implantation of radium in the cervix and by deep x-ray therapy may prove to be the most successful method. The frequent extension of the neoplasm toward the bladder will complicate the separation of the vesicouterine culdesac and present the risk of incising a lower segment which is edematous, infiltrated, inelastic, and which does not furnish the passage that is necessary for a successful delivery.
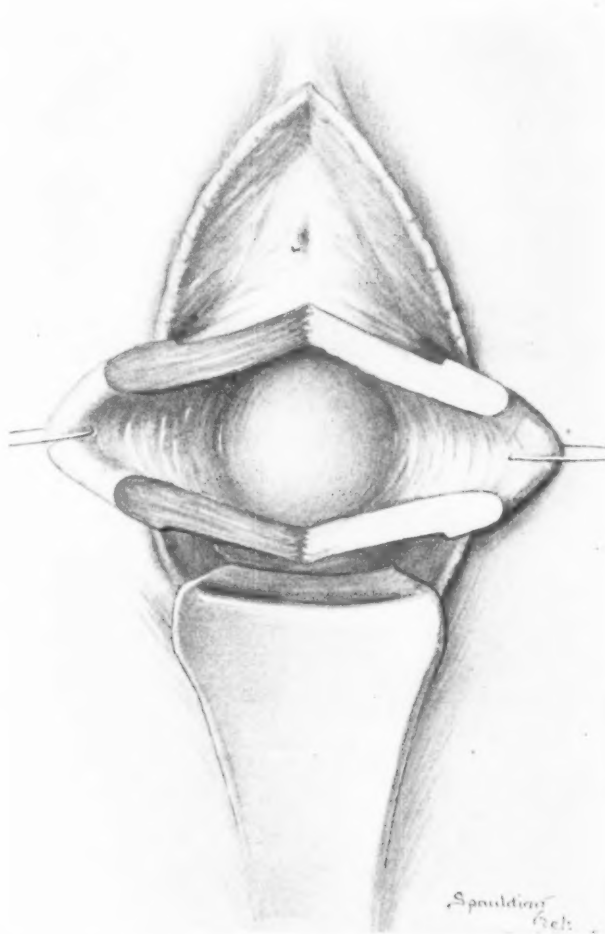
Fig. 4.—Anterior and posterior cervical incisions have been made. The delivery and extraction of the placenta and membranes follows.

Group II.—Lesions of the heart, lungs, and kidneys are still looked upon as suitable indications. As far as eclampsia is concerned, opinions are divided, but most obstetricians, nowadays, favor the Stroganoff conservative treatment or some modification of it, rather than the emptying of the uterus during convulsions. The vaginal cesarean section, nevertheless, is of marked value in the toxemic pa-

tient who fails to improve under the conservative treatment and when convulsions are imminent, since most gravidae delivered before the onset of convulsions have a much better chance of recovery. Vaginal hysterotomy is one of the accepted methods of terminating pregnancy in such cases. Premature separation of the normally inserted placenta in its milder forms and where no cervical dilatation exists may be treated successfully by vaginal cesarean section, but since we recog-
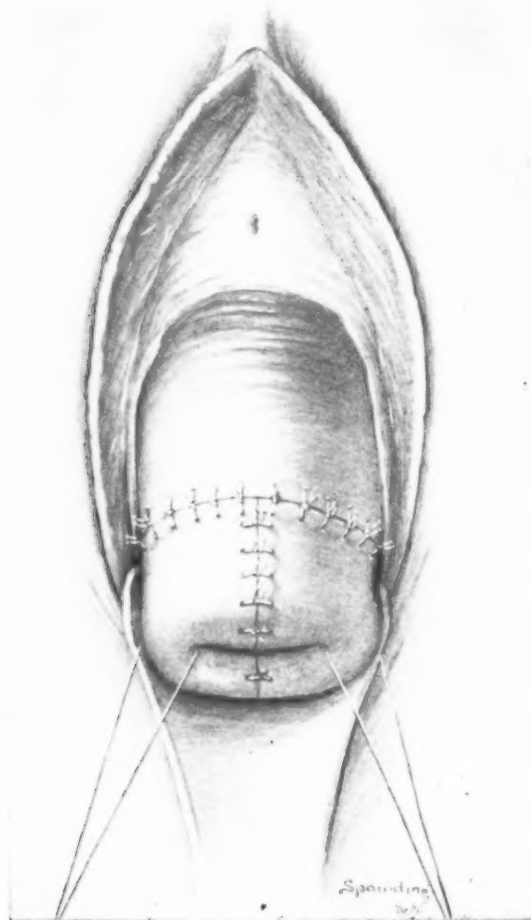
Fig. 5.—The cervical incisions have been closed with interrupted sutures of No. 2 chromic catgut, likewise the edges of the vaginal incisions.

nize more and more the association of uterine apoplexy in the severe forms of this condition, abdominal delivery with the conservation or the sacrifice of the uterus, depending upon the extent of the lesion, seems to be the better form of treatment.

Group III.—When the mother is dead or dying, I personally can see no reason for doing an operation, the technic of which is as complicated

as vaginal cesarean section, when the abdominal method which is quicker and simpler cannot in any way jeopardize the mother's chances and will certainly do a great deal more for the baby, the only reason for operating in this type of case.

Group IV.—When only the infant is in danger from a slow labor and from compression of the cord, vaginal cesarean may be indicated if the child is premature. At full term, the abdominal section is the preferable operation of the two.

Placenta previa is one of the obstetric complications for which vaginal hysterotomy has been recommended. The literature shows that this procedure has been employed, with success, in a number of cases of low implantation of the placenta. However this may be, the majority of operators would not agree that placenta previa is an indication for vaginal cesarean section for the following reasons: the hemorrhage of placenta previa added to the blood loss which may occur during the operation might well enough be fatal; the operation is not as rapid and as simple as abdominal section, and lastly, the hemorrhage from placenta previa would so obscure the field as to add materially to the difficulties of the procedure. In view of these reasons abdominal cesarean section should be chosen in central and in some cases of partial placenta previa, and a more conservative method, such as the insertion of a Voorhees' bag, or Braxton-Hicks version in the marginal variety.

Vaginal hysterotomy may be performed at the full term of pregnancy. I have personally so delivered a woman who had reached term with a good result; however, it offers no advantages over the low cervical cesarean section with a mature child, and I feel now that after the eighth month of pregnancy the large fetal head may extend the incisions and add materially to the risk of injuring the bladder. For this reason, it is my custom to resort to the abdominal cervical cesarean after the eighth month of pregnancy when an indication for immediate delivery arises.

A previous cervical cesarean section may seriously complicate the technic of the vaginal operation, since the bladder is much more adherent than in the normal condition. The anterior peritoneal culdesac having been obliterated, a definite interference with the rising of the bladder in the abdominal cavity exists during delivery and thereby predisposes to the laceration of this organ by the fetal head.

Vaginal cesarean section, therefore, finds its largest field of usefulness when an indication for the rapid emptying of the uterus arises up to the end of the eighth month of pregnancy, in:

1. Lesions of the heart, lungs, and kidneys.

2. Toxemia of pregnancy in the presence of impending convulsions and in gravidae who did not improve under the conservative treatment.

3. Pernicious vomiting of pregnancy in a gravida who is dehydrated and exhausted and who would not stand a long labor.

4. Premature separation of the normally inserted placenta in its milder forms.

TECHNIC OF OPERATION

A weighted speculum is introduced in the vagina to expose the cervix. The cervix is brought down by means of two volsellas, one at each lateral angle, and dilated with Hegar dilators to assure drainage. A suture of chromic catgut is placed at each lateral angle of the cervix and left long. These sutures facilitate the bringing down of the cervix after delivery. If the cervix is not readily brought down by this method, a No. 4 Vorhees' bag is introduced into the uterus; it is filled with 1 per cent lysol solution, and is used as a tractor. A transverse incision is made in the anterior vaginal wall at the point of attachment of the bladder to the cervix, and the bladder together with the vaginal wall is dissected upward to the reflection of the anterior peritoneal culdesac. The bladder is never separated from the vaginal wall, as is illustrated in most textbooks, because this has been found unnecessary and predisposes to bleeding and injury to the hyperemic and friable vaginal wall.

The bladder is now held under the symphysis by means of a wide retractor, and the anterior lip of the cervix is incised up to the vesicouterine culdesac, care being taken not to enter the peritoneal cavity. The posterior lip of the cervix is pulled upward toward the symphysis, with volsella or the bag, as the case may be, and a transverse incision is made at the junction of the vagina and the cervix. The posterior culdesac and the rectum are reflected downward, and the posterior lip is incised as far up as possible, care being taken to respect the abdominal cavity.

The instruments are now removed and the hand is introduced into the uterus, an internal podalic version and a breech extraction are performed in the usual way, and the placenta and membranes are extracted manually. By injecting pituitary extract and an aseptic preparation of ergot, the bleeding is usually controlled, so that packing is rarely necessary.

The cervical incisions are closed with interrupted sutures of No. 2 chromic catgut; the vaginal incisions are sutured in the same manner with the same material.

When the fetus is small, it is not necessary to incise the cervix posteriorly, as the anterior incision usually gives enough room for its extraction.

AUTHOR'S SERIES OF VAGINAL CESAREAN SECTIONS

TABLE I. NUMBER OF PREGNANCIES

Para i	17
Para ii	2
Para iii	5
Para iv	2
Para v	1
Total	27

TABLE II. PERIOD OF GESTATION

Full Term	1
8 months	4
7 1/2 months	3
7 months	11
6 1/2 months	4
6 months	1
5 1/2 months	1
5 months	1
4 months	1
Total	27

In the four cases where the period of gestation had not reached six and a half months, or the period of viability, the fetuses were macerated. In the case of one of the gravidae the pregnancy had been carried the fullterm, although she was delivered of a five months' macerated fetus.

TABLE III. INDICATIONS

Toxemia of Pregnancy, Convulsive Type	6
Toxemia of Pregnancy, Nonconvulsive Type	5
Toxemia of Pregnancy, Pernicious Vomiting Type	3
Toxemia of Pregnancy, Ablatio Placentae	2
Toxemia of Pregnancy, Pyometra-Rigid Cervix	1
Toxemia of Pregnancy, Previous Nephrectomy—Retained macerated fetus, Eighth month of pregnancy	1
Chronic Nephritis and Pregnancy	1
Pulmonary Tuberculosis, Advanced	1
Mitral Stenosis and Regurgitation, Pyelitis	1
Congenital Pulmonary Stenosis	1
Hydramnios, Rigid Cervix	1
Rigid Cervix, Long Labor	2
Pyelitis	1
Attempts at delivery before admission for hemorrhage, 6½ months of Pregnancy. Dead Fetus. Contracted Uterus	1
Total	27

TABLE IV. INDUCTION OF LABOR BEFORE VAGINAL CESAREAN SECTION

Induction with the Voorhees bag	5
Induction with cervical and vaginal gauze pack	1
Induction with bougies	1
Total	7

In these 7 cases, although contractions started, the cervix did not dilate, and as the patients became gradually worse, the labor was terminated by vaginal hysterotomy.

TABLE V. CERVICAL INCISIONS—EPISIOTOMY

Anterior Incision	12
Anterior and Posterior Incisions	15
Episiotomy performed 5 times	

TABLE VI. MATERNAL RESULTS

Maternal Recoveries		26
Maternal Death		1
Maternal Mortality	3.7%	

TABLE VII. FETAL RESULTS

Macerated Fetus		9
Stillborn Fetus		8
Lived 24 hours or less		6
Lived one month		1
Now alive		3
Total		27
Total fetal Mortality	88%	
If we exclude the 9 macerated and 8 stillborn fetuses	37%	

Naturally these infants with one exception were markedly premature and toxic.

TABLE VIII. PELVIC DELIVERIES FOLLOWING VAGINAL CESAREAN SECTION

1 patient had a normal delivery in the hospital.
1 patient had a normal delivery in her home by another obstetrician.
1 patient had a version and breech extraction after twelve hours of labor and subsequently a normal delivery.
All these mothers and children are well.

In this series of twenty-seven operations, one mother died. Mrs. A. K., para i, thirty-one years of age, was seen for the first time on April 27, 1919. She had been admitted to a hospital during a severe hemorrhage due to the premature detachment of a normally inserted placenta. The cervix was not taken up, and there was no dilatation. Her pulse was 120 in rate, thready, and of poor quality. The vertex was presenting in left occipitoanterior position. An immediate abdominal delivery was decided upon on account of the hemorrhage and the long rigid cervix without dilatation. On opening the peritoneal cavity, a rent about two inches long running obliquely from the fundus toward the right tube was noticed; this extended through the serosa and muscularis, but not through the mucosa. There was considerable blood, both free and clotted, in the peritoneal cavity. The uterus was incised in the median line, there being practically no bleeding from the cut uterine wall. The placenta was almost entirely separated. A small stillborn male fetus, at term, was extracted by the breech; the placenta, membranes, and clots were removed, and the cervix was dilated from above. The uterine incision was sutured in two layers with No. 2 chromic catgut. The rupture in the uterus was repaired with figure of eight sutures of the same material. No attempt was made to remove the blood or fluid from the abdominal cavity, and the abdomen was closed in layers.

She had an uneventful convalescence and was discharged from the hospital on May 23, 1919, on the twenty-sixth day after operation.

Her second pregnancy evolved normally and on March 28, 1921, she was delivered of a normal female child, presenting by the breech in right sacroanterior position, by a low cervical cesarean section. At this time it was found that the previous incision and the rent in the uterus was well healed.

The patient was in poor physical condition throughout her third pregnancy. She had reached the full term, although her uterus corresponded in size with a five months' pregnancy. On August 16, 1924, she developed uterine hemorrhages and a septic purpura. She had shown signs of toxemia during the whole of her pregnancy. As she was failing rapidly, it was thought that her only chance rested in quickly emptying the uterus. This was done by vaginal cesarean section on August 16, 1924. Pyometra was found to be present, half a liter of foul pus being evacuated from the uterus as well as a macerated, five months' female fetus. There was no difficulty with the operation, which was rapid. The patient was returned to her bed in poor physical condition. She died three hours later from shock, toxemia, and severe uterine infection.

CONVALESCENCE

Of the twenty-six mothers who recovered, twenty-five had a satisfactory puerperium. The twenty-sixth puerpera made a good recovery, but her convalescence was complicated by a vesicovaginal fistula, which is still present and which I hope to repair in the near future. The history of her case follows:

Mrs. M. R., a primipara, twenty-eight years of age, was admitted to the Gynecological and Obstetrical Service of the Carney Hospital on November 25, 1923, as an emergency case. Her family history showed nothing abnormal. Her previous history elicited that she had had measles, pertussis, and influenza. Her menstrual history was normal. On admission to the hospital at 3:30 P.M., it was found that she had been in labor for forty-eight hours. She complained of headache, nausea and vomiting, marked swelling of her hands and feet, and shortness of breath. She had been examined vaginally in her home a number of times; the exact number of examinations could not be ascertained.

On examination, the heart showed the typical signs of pulmonary stenosis, that is, a thrill and a systolic murmur heard to the left of the sternum in the second intercostal space; the pulmonary sound was replaced by a diastolic murmur; there was hypertrophy of the right heart and marked edema of the bases of the lungs. Considerable edema of the extremities existed, otherwise the physical examination was negative. She was pregnant at term, with the vertex presenting in right occipitoposterior position. On rectal examination, the cervix was found to be two fingers dilated, and the fetal head was overriding the symphysis. The patient was exhausted, and her condition looked desperate. She was delivered by a low cervical cesarean section, under morphine-scopolamine local anesthesia, of a living female child, weighing eight pounds and one ounce.

The convalescence was surprisingly simple; she had a temperature of 101° F. on the tenth postoperative day; otherwise the temperature remained below 100°. The highest pulse rate was 108. She was discharged on the fifteenth day after operation.

She was followed at the prenatal clinic of the Carney Hospital during her second pregnancy. Her confinement was expected on April 8, 1926. She complained of severe dyspnea, was markedly cyanosed, and had to sleep in a chair, as it was impossible for her to lie down. She entered the service on March 9, 1926, in what appeared to be a very serious condition. She had all the signs of extreme cardiac decompensation. On March 11, 1926, she was delivered of a male child weighing seven pounds, by a second low cervical cesarean section under scopolamine-morphine local anesthesia. Her cardiac condition gave considerable alarm during the puerperium, but her temperature never rose above 101° F. She had a sinking spell on March 28, and hopes for her recovery were almost abandoned. Nevertheless, she was discharged on April 4, 1926, on the twenty-fourth day after operation.

She showed signs of cardiac distress very early in her third pregnancy, but with rest in bed and proper medical treatment her gestation reached the seventh month. On May 19, 1927, she collapsed in the clinic where she had reported for a prenatal visit and was immediately sent to the hospital where she was kept in bed until May 23, 1927, the day of operation. Since her cardiac reserve was very low, it was thought that delivery by vaginal cesarean section would offer a better chance than would an abdominal delivery, since the added shock of opening the peritoneal cavity, slight though it may be, might result fatally. She was, therefore, delivered by vaginal hysterotomy under spinal anesthesia, anterior and posterior incisions being made in the cervix. There was no difficulty in separating the bladder. The delivery was accomplished by means of version and extraction of a stillborn male child. The head was soft and showed early signs of maceration. The bladder was injured during the delivery but was repaired immediately. A Pezzer catheter was introduced, and she was returned to her bed in fair condition. She convalesced satisfactorily, except for the fact that a vesicovaginal fistula soon developed. She was discharged from the hospital on the forty-fourth day after operation, her long stay being due to her cardiac condition. At the time of her discharge an x-ray of her heart was taken, and this showed marked dilatation of the right auricle. At this time the bladder held eight ounces of urine, and the vaginal leakage was slight. She was again admitted to the hospital on June 23, 1927, because of her heart condition and was again discharged eight days later. There was no change in the condition of the fistula. She has been examined every two weeks since that time and an attempt to close the fistula will be made later.

CONCLUSIONS

1. Vaginal cesarean section is a useful operation when an indication for immediate delivery arises in a gravida with a long, rigid, undilated cervix, up to the end of the eighth month of gestation.

2. The operation may be done at term, as shown by one of the cases of this series; but here the difficulties are greater, and there is danger of the incisions tearing in the peritoneal cavity because of the large size of the child.

3. A previous low cervical cesarean section complicates the technic of the operation, since the anterior peritoneal culdesac has been obliterated. This may predispose to injury to the bladder during delivery.

4. Since the operation is extraperitoneal, postoperative complications are negligible.

5. The puerperium, as a whole, resembles that of any operative pelvic delivery.

6. The low transperitoneal abdominal cervical cesarean section which offers nearly as much protection against infection as does the vaginal hysterectomy has displaced the latter operation in a number of clinics because of its simpler technic.

270 COMMONWEALTH AVENUE.

IMMEDIATE REPAIR OF BIRTH CANAL INJURIES FOLLOWING DELIVERY

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THE care of the woman who has given birth to a child, is so different today from what it was thirty years ago that it may be interesting to state the reasons for certain present-day procedures and the benefit that is supposed to be derived from their use.

Every effort possible is now made to obtain rapid and permanent involution of the uterus.

Subinvolution of the uterus is the cause of many of the ailments following childbirth. Conditions that invite subinvolution of the uterus are, therefore, the ones we are now trying to overcome and avoid, such as needlessly prolonged second stages of labor due to faulty positions of the child and not recognized; dystocia, maternal and fetal; prolonged pressure and stretching of the tissues of the birth canal, resulting in rectoceles and cystoceles; lacerations of the vagina, perineum, and lower uterine segment, all of which have a tendency to produce what we term subinvolution.

For the past few years we have heard, from various parts of the country, about the different methods used by many men to overcome these

tendencies, and we have carried on some experiments along these lines, which have proved beneficial to the patients.

Various exercises while the patient is in bed, beginning as early as the second day after delivery; changes in posture; the Fowler position for drainage, and later the knee-chest position to prevent retrodisplacements and for the purpose of emptying the larger veins in the pelvis, thighs, and abdomen, thereby avoiding varicosities and perhaps lessening the tendency to thrombosis in these veins with their resultant bad effects and postpartum displacements of the uterus, which are often permanently corrected. Patients get out of bed feeling stronger, and have less of that dragged feeling with backache

Fig. 1.—Patient in position for examination after third stage of labor.

which was formerly so common. Constipation is less, and there is better control of the bladder.

The first step in my efforts to obtain good involution begins in properly preparing the patient for delivery. No patient should be delivered with a full bladder. The bladder should be emptied with a catheter before delivery, and if there is time, an enema may be given to cleanse the lower bowel.

I believe that the attendant is *not* responsible for lacerations of the lower uterine segment or cervix following delivery, unless he has needlessly interfered, or has dilated manually, or has applied forceps before complete effacement of the cervix has taken place, or has attempted version and extraction through an undilated cervix.

Many times I have examined carefully the lower uterine segment and the cervix and os following normal, untouched cases, which were delivered as vertex cases, and I have been surprised at the damage done as compared with those in which intelligent interference was

used; from these observations I believe that it is necessary, in order
to obtain the best results, to prepare properly the birth canal for
delivery.

The statement, previously made, that the attending physician is not
always responsible for tears and injuries to the lower uterine segment
of the uterus, does not apply so generally to the vaginal canal, as
lacerations of the vagina and perineum can be prevented and should
be greatly reduced in numbers and degree, when certain lines of pro-
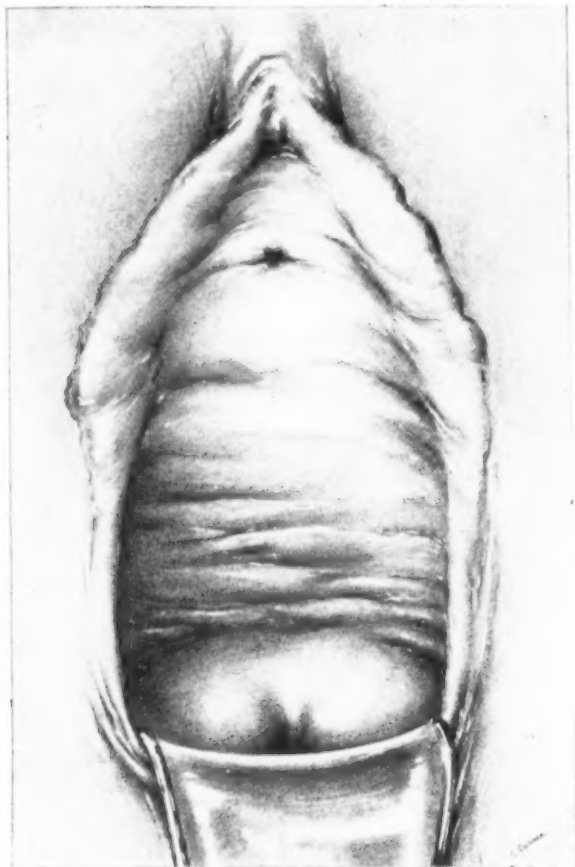
Fig. 2.—Birth canal exposed by speculum.

cedure are carried out. In order to accomplish these desirable ends,
the patients, after the first stage of labor is finished or during the
early part of the second stage, should be given chloroform to the
surgical degree. No such term as obstetric anesthesia is used by me.
A process of dilating or ironing out of the vaginal canal is begun by
first introducing into the vagina one finger and beginning pres-
sure from within out and from above down, then two fingers are in-
serted and then three fingers and finally the whole hand is introduced,

using green soap as a lubricant. Soap is not only used as a lubricant but is a cleanser for the canal. This preparation is carried out in every case, no matter what the position of the child is or what manner of delivery is to be used, as it reduces to a minimum the possible damage to the soft parts. By such a procedure my patients are not shocked, the elasticity of muscle is not destroyed, facia is not so often broken, allowing rectoceles and cystoceles to occur; and episiotomies are unnecessary. The danger of hemorrhage need not be feared, pro-
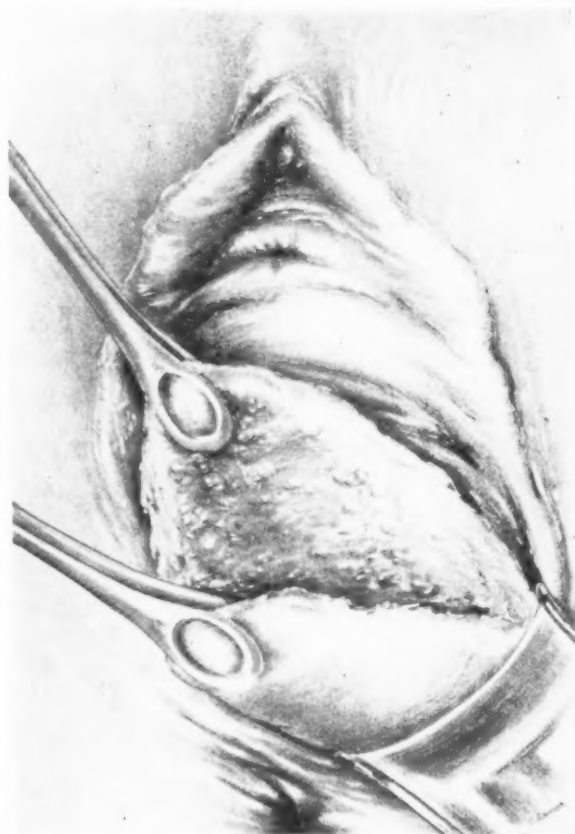
Fig. 3.—Lower uterine segment brought down for inspection.

vided proper management of the third stage of labor is carried out, that is, in not hurrying in the delivery of the placenta and membranes. If the case be delivered as a vertex, time should be allowed for the passage of the head through the vaginal canal, and extension of the head should not be allowed to take place until the occiput is well under the symphysis. When the head is finally delivered as far as the ears, the operator should grasp the occiput firmly and rotate the head to one side or the other, according to the position of the child, and instead of the nose and chin passing over the thinned perineum, they

will be delivered sideways, and only the smooth side of the head will pass over the thinned perineum, thereby avoiding tears produced by the uneven surface of the face. After the head is delivered, it is allowed to assume its natural position of face down.

In the event of forceps delivery—I am speaking now only of mid forceps or low forceps, believing that high forceps operations, so-called, are things of the past—great care should be used in first making proper application of the forceps; then the head should be
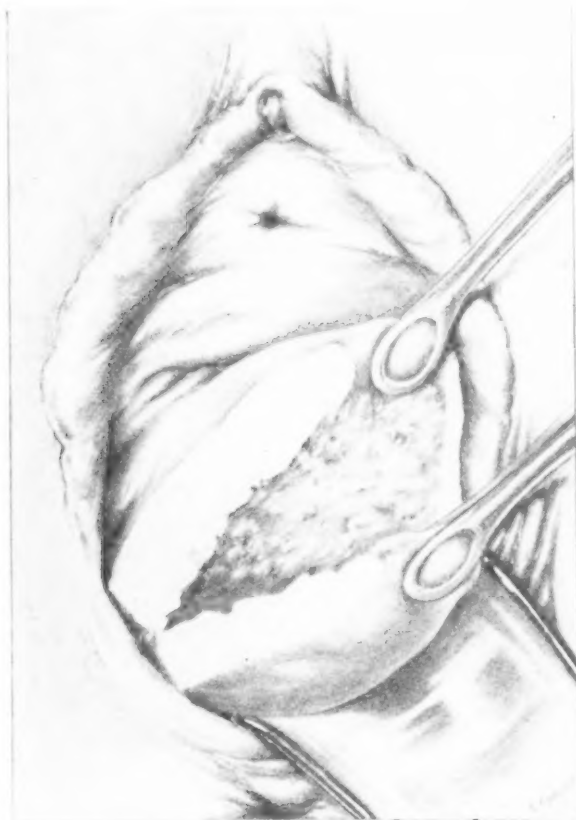
Fig. 4.—Beveling edges of cervix.

carefully brought down sufficiently for the instruments to be removed, when the head is delivered without them, exercising the same care to avoid lacerations as one would in a spontaneous vertex case.

In version and extraction, the same procedure must be used—first, to iron out thoroughly the vaginal canal until all resistance is overcome; it sometimes requires twenty or thirty minutes to accomplish this. I always wear long rubber gloves in my work, and I introduce the whole left hand into the vagina after it has been dilated, with the palm up, carrying the hand between the membranes and uterine

wall, if the membranes have not ruptured, well up toward the fundus
of the uterus, separating the membranes around as far as the pla-
centa, and avoiding, if possible, loosening the placenta, as that in-
vites hemorrhage. The membranes are ruptured high up so as to save
as much as possible of the amniotic fluid and the arms are now
folded across the chest, if they are not already in that position, to
avoid the complication of the extended arm, the feet are brought down
and delivered, both at the same time between the first and middle
fingers of the operator's left hand. From now on the extraction
proceeds slowly and carefully to avoid damage to the soft parts of
the mother and also to avoid injury to the child from too rapid ex-
traction; the knees are now delivered and the buttocks rotate to the
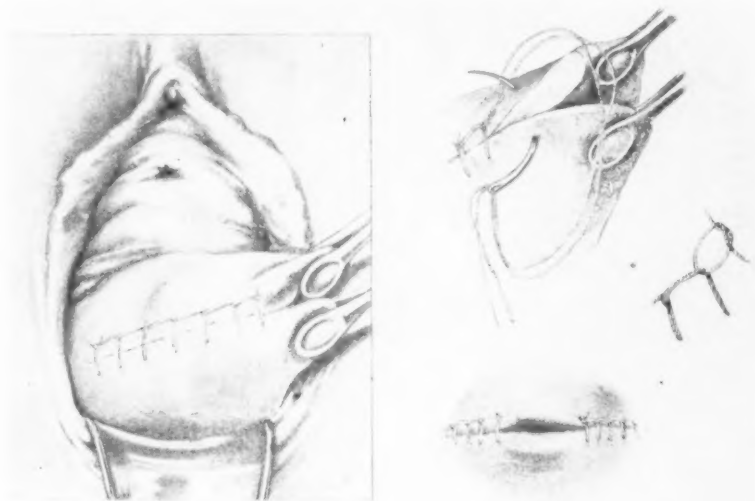
Fig. 5.—Showing insertion of sutures and external os.

hollow of the sacrum, where time is taken to allow the abdomen of
the child to adapt itself to the pelvic outlet; then by gentle traction
the back is rotated forward, squarely under the pubic arch, and trac-
tion is continued until the lower angle of the scapulae are seen, when
the finger of the operator is put in the posterior axillary fold and the
shoulder is rotated forward under the pubic arch. The child is then
supported on the operator's hand and the posterior shoulder is ro-
tated in the same manner and delivered as an anterior shoulder. Trac-
tion is not made downward toward the floor but in the direction of
the patient's thighs, which are in a modified Walcher position.

The delivery of the after-coming head is now made as a flexed head
by placing the fingers of the operator's left hand on the chin of the
child and making pressure on the woman's abdomen above the sym-

physis with the right hand, thereby pushing rather than pulling the head through the pelvis. Great care should be taken to preserve the soft parts of the mother by not using too much haste in the delivery of the head. The child's body should still be in the direction of the mother's thighs and not bent back over the abdomen.

Breech cases, after complete effacement of the cervix, are delivered as footling cases, forceps being used upon the after-coming head if necessary.

For all of this work complete anesthesia is necessary, and damage to the vaginal canal and perineum will be reduced to a minimum.

After completion of the third stage of labor and while the patient is still under the anesthetic, careful inspection of the lower birth canal is made to see whether any repairs are necessary; this is accomplished by bringing down the anterior and posterior lips of the cervix with two cervix forceps. This is where I believe that great benefit can be done the patient by opening any cysts in the cervix or by removing them either by a broad sharp curette or with flat scissors, and also any diseased tissue that may be present. It is by this procedure that I cure the cases of old leucorrhea by beveling out the cervix and bringing the new-made raw edges together with two to three interrupted sutures of catgut or by a continuous suture starting at either angle and going toward the center, leaving space enough to admit easily the cervix forceps and thus not prevent subsequent drainage.

The lacerations of the cervix are unilateral, bilateral, and stellate. The unilateral tears are the most common and are found to some degree in practically every case and may be so slight as to require no attention; this is especially so in the primipara. The bilateral tears are found more frequently, following prolonged forceps and so-called dry labors. The stellate is the least common of all and depends largely upon the cervical tissue in which direction the laceration extends.

I have found that unilateral tears appear on the side of the cervix where the occiput has remained longest. For example, in prolonged L.O.P. positions it would appear on the left side, and in R.O.P. positions on the right side, being further proof of the damage resulting from these posterior positions.

In my work I do not often see bleeding from the tears of the cervix. Bleeding, if it occurs, usually comes from the placental site in the uterus and not from the cervix, proving that extensive and deep tears high up involving this circular artery are not common. Sutures are not always placed on account of visible lacerations, but many times in primipara they are put in the angle of the cervix to aid involution. In cases where previous lacerations existed, enough tissue is removed to bring the raw edges of the cervix together so as to cover all everted

surfaces. It is not always necessary to remove all of the scar tissue, as this is so stretched out and softened that future absorption of it takes place during the subsequent involution.

I am satisfied that the damage done the lower uterine segment is far less in cases that have been manually delivered than in many of those that are left to nature. The lower uterine segment, when brought down for inspection, is a sorry spectacle at best, resembling more the appearance of a ploughed field than anything I can describe, but it is far less rough in appearance in cases where intelligent interference has been made than in those cases left to nature.

In those cases not assisted, a dark congested area, resembling a band from $\frac{1}{8}$ to $\frac{1}{4}$ of an inch in width, is always noticed encircling the lower uterine segment and sometimes sufficiently devitalized so as to slough and cause future morbidity. This ring is never seen in the cases delivered manually.

I never have seen strictures of the cervical canal following this work. Drainage from the uterus is always ample and a postpartum examination from six to eight weeks after delivery shows no narrowing of the canal.

For the past three years I have used this method with very gratifying results in more than 1000 cases. I have found that it cures old previously existing cervicitis; it repairs old cervical lacerations, and it is a great aid in accomplishing and maintaining involution.

In primiparae, as a rule, no tissue is removed and sutures are merely placed to bring the torn edges together, thereby reducing future scar tissue to a minimum.

In old multiparae near the menopause, no attempt is made to save the cervix. I curette or cut down until I reach healthy tissue, and then bring the edges together in the manner heretofore described. This does away with all previous cervical erosions and cures, as near as anything will, the cervicitis which was due to the infection of the cervical glands, and can only be reached at this time and in this manner; this renders treatment with the cautery afterwards unnecessary. I never have seen any bad effects in subsequent labors. It does not delay the patient's stay in the hospital, in fact, if anything, it lessens the stay by bringing about a more perfect and speedy involution.

Patients return for final examination in six weeks, and many times it is difficult to say whether or not a full-term child had been delivered through that canal. At this time a sound can be introduced into the uterus to see that a proper canal exists, and if necessary, it can be dilated while the tissues are soft and yielding. Tears in the vaginal canal and perineum should always be repaired immediately. If such an unfortunate result as a complete tear into the bowel occurs, immediate repair should be attempted in the hope that possibly a good

result will be obtained. Failing in this, no further attempt at repair should be made until lactation ceases and menstruation appears, as then proper operative procedures can be better carried out.

This work which I have been engaged in for some years has also been done by Dr. J. L. Bubis, of Cleveland, and is reported in an article in THE AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY, August, 1925. He, however, extends the procedure and includes the repair of old cervical lacerations and the repair of cystoceles and rectoceles and has operated upon 152 cases very successfully. However, I am inclined to believe that there is some risk in this extensive work and not enough accomplished in securing involution; perhaps a better repair can be brought about later, but to neglect the repair of an old lacerated cervix with all of its associated pathology is wrong in the light of our present knowledge, because a proper involution of the uterus can never be brought about where there is an old diseased cervix. Furthermore, by this timely procedure we protect our patients from future malignant disease of the cervix, which in a certain percentage of cases begins at the site of a cervical injury, and we relieve them of the expense of a second operation, some pain and suffering, and some danger from a second anesthesia.

689 FOREST AVENUE.

THE ETIOLOGY AND TREATMENT OF THE BLEEDING UTERUS

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INTRODUCTION

THE recognition of the disease causing or accompanying a uterine hemorrhage is of the greatest importance for the selection of the indicated method of treatment. A perusal of the medical literature on this subject depicts the differences prevailing in the interpretation of the underlying causes, the diagnosis, and the methods of treatment. For instance some of the advocates of either endocrinology, or surgery, or radiation therapy, leave the impression that all the varieties of uterine hemorrhages may be entirely combated by the sole use of one of the methods. The points at issue can only be cleared by a study of the causative factors and the functional and pathologic changes associated with uterine hemorrhages. Thereby a basis may be constructed whereon to build correct methods of prevention and of cure.

The investigations carried on by Fraenkel,[1] R. Meyer,[2] Novak,[3] and others on the internal secretion of the ovary and the influence of the graafian follicle and the yellow body on menstruation; the studies of

Hitschmann and Adler,[4] R. Schroeder,[5] and others on the cyclic changes in the histology of the endometrium, as well as the researches of Zondek and Aschheim,[6] Biedl,[7] Allen and Doisy,[8] and others on the influence of disturbances in the endocrine system on uterine and ovarian function and the composition of the maternal blood have greatly advanced our knowledge of the processes of ovulation and menstruation and their disturbances. The development of modern surgery, the introduction of x-rays and radium in therapy, and the standardization of endocrine extracts have all contributed to progress in the treatment of uterine hemorrhages. I shall endeavor to classify the causes of uterine hemorrhages according to the underlying pathologic physiology and histology and discuss the indicated methods of treatment for each group.

DEFINITION AND CLASSIFICATION

Uterine hemorrhages have been divided into (1) those dependent upon recent pregnancy, and (2) those independent of recent pregnancy.[9] It is now generally conceded that uterine hemorrhages occurring without the periods of gestation, pregnancy, and early puerperium are caused by either a disturbance in the uterus or the ovary, or internal diseases, especially of the endocrine system.

The gynecologic department in the medical school and hospitals, in which the material for study was obtained, is sharply separated from the obstetric department, and hemorrhages due to accidents of pregnancy, labor, and puerperium are rarely seen. They, however, constitute a large number of cases of genital bleeding. Therefore, it is imperative that the possibility of such disturbances must always be borne in mind to prevent mistakes of diagnosis and treatment. If a woman during the childbearing period of life complains of amenorrhea or a genital hemorrhage taking place after a period of amenorrhea or a missed menstrual flow, she should be deemed pregnant until proved not to be pregnant. If one is in doubt, a waiting attitude and observation will help in the diagnosis.

A small number of uterine hemorrhages are caused by general internal diseases as hypothyroidism, hyperthyroidism, pituitary gland disturbances, thrombopenic purpura hemorrhagica, and atypical purpura, acute infectious diseases as influenza, typhoid fever, scarlet fever, variola, rheumatic fever, malaria, etc., decompensated cardiac lesions, pulmonary emphysema, hepatic disease, chronic nephritis, etc. Such uterine hemorrhages are seen by us only in consultation with the attending internist. These hemorrhages if erroneously assigned to the gynecologic department are grouped in the accompanying tables in the columns on general stasis, systemic diseases or unclassified cases. Heyn[10] reported 285 cases of menorrhagia and found that 11.85 per cent resulted from passive congestion due to

general diseases. In 791 cases of uterine hemorrhages reported below the instance of general disease was 21 or 2.65 per cent.

A most careful interrogation of the patient is a *sine qua non* for the correct clinical interpretation of the uterine bleeding. The use of hemorrhage charts as devised by Kaltenbach[11] and R. Schroeder[12] is a great help to determine the type of bleeding, particularly to the medical student and the general practitioner, who see these cases usually before they enter a gynecologic clinic.

Menstrual hemorrhages are termed menorrhagias; intermenstrual hemorrhages are designated metrorrhagias. Menorrhagia implies that the menstrual flow is either too profuse, lasts too long, or occurs too soon in comparison to the behavior and type of the process of menstruation normal for a given case. The profuse and prolonged menstrual flow has also been termed hypermenorrhea; the too frequent menstruation, polymenorrhea. Both types may occur at the same time. The amount and duration of the menses depend upon the functional state of the uterus and active or passive pelvic hyperemia. The factors controlling the amount and duration of the menstrual flow are the contractility and tonicity of the uterine muscle, the degree of engorgement of the pelvic blood vessels, and the coagulability of the blood. Loss or decrease of contractility and tonicity is associated with hypoplasia, general and uterine asthenia, and myomas. Hyperemia may be either active or passive. Active hyperemia results from infections and displacements of the genitals, and passive hyperemia is caused by cardiac, pulmonic, hepatic, and nephritic diseases.

The periodicity of the menses depends upon the time duration of ovulation and the life duration of the expelled ovum. When the ovum succumbs, the menstrual decidua becomes detached, menstruation is started, resolution of the yellow body ensues and another graafian follicle develops. The duration of development of the graafian follicle is about fourteen to eighteen days, hence the potential life span of the expelled ovum is probably ten to fourteen days. If the time period of maturation of the follicle is shortened, or if the expelled ovum possesses a low vitality and is short-lived, then the menstrual flow will come on too soon. Disturbances of ovarian function may be primary or secondary. The primary dysfunctions are associated with the congenital hypoplastic state, which also involves the ovaries. Ovarian function suffices to bring about maturation of a follicle, but the produced ovum has a lessened viability or vitality. Such polymenorrheas occur with greatest frequency at the beginning of menstrual life when ovarian function is in the process of development to maturity, and at its termination when ovarian activity begins to ebb. The former are termed pubescent; the latter are designated menopausal hemorrhages.

Secondary ovarian hypofunction is caused by infections which invade the ovary or the corpus luteum, and endocrine disturbances and general diseases which interfere with the functions of the ovary.

Metrorrhagias are mainly caused by a chronic hyperplasia of the endometrium—the result of the persistence of a corpus luteum, malignant diseases involving the uterus, senile cervicitis and vaginitis, lues, etc.

STATISTICS

A study of 2523 consecutive gynecologic cases, observed during 1924 and 1925, was made. At the Cook County Hospital 804 cases, and at the Mercy Clinic Hospital 1719 cases were seen. Of the latter 130 cases had to be subtracted due to insufficient data. The frequency of uterine hemorrhages was 42.70 per cent in the former and 28.07 per cent in the latter institution. (Table I.) This difference made it desirable to classify the hemorrhages separately for each hospital.

In Table II it is seen that inflammations of the uterus and tubes furnish the largest numbers of hypermenorrheas, namely, 51.04 per

TABLE I. GROUPING OF CASES ACCORDING TO TYPE OF BLEEDING

	COOK COUNTY	MERCY	TOTAL
Number of cases investigated	804	1589	2393
Number of bleeding uteri	344	447	791
Per cent of frequency	42.70	28.07	33.06
A. Menorrhagia			
Number	247	210	457
Per cent	72.04	46.98	57.78
a. Hypermenorrhea			
Number	223	163	386
Per cent	64.77	36.47	48.55
b. Polymenorrhea			
Number	24	47	71
Per cent	7.27	10.51	9.22
B. Metrorrhagia			
Number	97	237	334
Per cent	27.96	53.02	42.23

TABLE II. GROUPING ACCORDING TO UNDERLYING DISEASES

Percentages Calculated for Each Type of Bleeding

A, a: Hypermenorrhea 386 Cases

	DECREASED CONTRACTILITY			HYPEREMIA	
	MYOMA	HYPOPLASIA	ASTHENIA	PELVIC INFECTIONS	GENERAL STASIS
Total number	131	17	38	197	3
Per cent	33.94	3.63	10.62	51.04	0.77

A, b: Polymenorrhea 71 Cases

	PRIMARY OVARIAN HYPOFUNCTION		SECONDARY OVARIAN HYPOFUNCTION	
	PUBESCENT	MENOPAUSAL	OVARIAN INFECTION	SYSTEMIC DISEASES
Total number	13	4	48	6
Per cent	18.31	5.63	67.71	8.45

B. Metrorrhagia: 334 Cases

	CHRONIC HYPERPLASTIC ENDOMETRITIS	MALIGNANCY	ACCIDENTS OF PREGNANCY	SENILE CERVICITIS	LUES	UNCLASSIFIED
Total number	42	202	52	5	13	20
Per cent	12.28	60.48	15.59	1.49	3.89	6.27

TABLE III. GROUPING ACCORDING TO UNDERLYING DISEASES
Percentages Are Calculated From Total Number of Bleeding Uteri

A. Menorrhagia

A. HYPERMENORRHEA

| | DECREASED CONTRACTILITY | | | HYPEREMIA | |
	MYOMA	HYPOPLASIA	ASTHENIA	PELVIC INFECTIONS	GENERAL STASIS
Mercy number	38	14	25	84	2
Per cent	8.50	3.13	5.59	18.79	0.45
County number	93	3	13	113	1
Per cent	28.10	0.84	3.78	32.21	0.28

B. POLYMENORRHEA

| | PRIMARY OVARIAN HYPOFUNCTION | | SECONDARY OVARIAN HYPOFUNCTION | |
	PUBESCENT	MENOPAUSAL	OVARIAN INFECTIONS	SYSTEMIC DISEASES
Mercy number	11	4	27	5
Per cent	2.46	0.89	6.04	1.12
County number	2	0	21	1
Per cent	0.45		6.11	0.28

B. Metrorrhagia

	CHRONIC HYPERPLASTIC ENDOMETRITIS	MALIGNANCY	ACCIDENTS OF PREGNANCY	SENILE CERVICITIS	LUES	UNCLASSIFIED
Mercy number	30	148	32	5	8	14
Per cent	6.71	33.11	7.18	1.12	1.78	3.13
County number	12	54	20	0	5	6
Per cent	3.46	15.70	5.60		1.40	1.68

cent; inflammatory diseases of the ovaries the largest number of poly-
menorrheas, namely, 67.61 per cent; and malignant diseases of the
uterus the largest number of metrorrhagias, namely, 60.48 per cent.
Among the colored women pelvic infections come first in frequency,
namely, 32.21 per cent, and myomas come second in frequency as a
cause for hypermenorrhea, namely, 28.10 per cent. The percentages
found in the other diseases give an idea of their relative importance.
(Table III.)

The higher percentages of menorrhagia in the Cook County Hospital
cases are due to the preponderance of myomas and pelvic infections.
About 80 per cent of these patients are colored which explains these
discrepancies. On the other hand the preponderance of the number
of metrorrhagias in the Mercy Hospital, admitting only white people,
is due to the greater frequency of malignancy.

TREATMENT

A patient suffering from uterine hemorrhages whether menorrhagia
or metrorrhagia, should not be treated unless the underlying cause
has been determined and a correct diagnosis has been made. All
bleeding that does not occur synchronously with the physiologic
menses must be viewed with grave suspicion until it has been proved
to be benign. If a nodule in the vaginal portion of the cervix is in-
cised and mucus exudes, it is probably benign; if arterial bleeding
ensues, then a diagnostic excision must be made, for the nodule is
probably malignant. If an ulcer on the vaginal portion of the cervix

is touched with a cotton applicator and does not bleed, it is probably benign; the cancer ulcer does bleed and the blood is arterial in character. If the introduction of a sound into the cervical canal and the uterine cavity, with strictest aseptic precautions, causes a thin stream of bright red blood to escape into the vagina, then this observation may be regarded as highly suspicious of malignancy. especially if the trickling of blood continues for some time after the manipulation. These signs are only a contributory means of arriving at a diagnosis and should not be conclusive. Carcinoma is a corrosive process; cancer tissue is friable, and these facts explain the observation. Every woman suffering from uterine hemorrhages should be examined and, if the cause does not become evident, the uterus should be curetted, suspicious looking nodules and ulcers of the cervix should be excised, and all the tissue should be examined microscopically. Such diagnostic curettage and excision of tissue should always be done immediately without any delay.

Postclimacteric bleeding without a lesion of the uterus is an important early sign of ovarian tumors. The tumors are often of the granulosa cell variety, and the uterine changes suggest ovarian hormonal influence. Such ovarian tumors are frequently carcinomatous. It would seem a mistake, therefore, to postpone operation until the appearance of a palpable tumor.

The principles of treatment of hypermenorrhea are: (1) the reactivation of the contractile power, (2) the tonicity of the uterine muscles, and (3) ablation of the active and passive hyperemia of the pelvis.

The polymenorrhea requires a correction of (1) the ovarian hypofunction, (2) the active ovarian hyperemia, and (3) any primary disease causing passive stasis.

The metrorrhagias indicate a removal of the underlying pathology.

Pelvic inflammatory diseases should be treated conservatively according to the well-established rules. If they do not react to conservative treatment, a temporary amenorrhea may be produced by the use of x-rays but not radium. A period of complete rest of the functions of the genital organs often contributes to a permanent cure.

Retrodisplacements and descent of the uterus should be corrected. The lacerated cervix should be repaired. The chronic cervicitis with erosions, hypertrophy, and nabothian cysts should be treated. If they resist treatment, the cervix should be amputated. The relaxed vaginal outlet should be restored by a perineorrhaphy, myorrhaphy, and anterior and posterior colporrhaphies.

The contractility of the uterus may be increased with hydrastis, extract of the posterior lobe of the pituitary gland, and adrenalin. Adrenalin solution 1:1000 may be injected intramuscularly in 0.75 c.c. doses twice weekly. Pituitary extract may be used in 0.015 gm. doses in solution intramuscularly twice weekly for twelve doses.

Hypoplastic uteri may be stimulated by the use of ovarian follicular hormone. Two hundred rat units should be injected intramuscularly twice weekly for about twelve doses. If results are not then obtained, the further administration is useless. Pratt and Allen[13] have reported clinical observations with injections of follicular hormone and state that they produce growth in the uterus.

Uterine asthenia is usually associated with general asthenia. As the body of the patient improves through regulation of diet, proper exercise, rest and sleep, and medication, so the tonicity of the uterus will increase.

Myomas, causing hypermenorrhea, may at first be treated conservatively. If a lasting improvement is not obtained, then surgery or radiation therapy are indicated. The contraindications to the use of radium and x-rays are so manifold that in thirty-eight bleeding myomas observed in this series at the Mercy Hospital, radium was indicated only seven times.

The polymenorrheas of puberty should be treated conservatively by general hygienic measures, hot vaginal douches, applications of heat with diathermy, the intramuscular injection at the beginning of the flow of 25 units of Collip's extract of the parathyroids, to be repeated within twelve hours. In the intermenstrual period 0.06 gm. doses of thyroid extract are given twice daily. If such measures do not bring results, then curettage is done, which is a stimulating measure as it promotes contractility of the uterus and thereby stimulates ovarian function. If the hemorrhages recur, curettage is repeated. The use of radium in pubescent women is deemed by us as absolutely contraindicated.

A more radical measure in both kinds of menorrhagia, due to functional disturbances within the uterus and ovaries, is curettage. Finally radium, x-rays or hysterectomy may be used, if all other measures have failed, and age does not contraindicate the use of rays. Curettage, fortunately, will arrest about 50 per cent of such bleedings. Radiation castration has supplanted corpus amputations for benign uterine hemorrhages in gynecologic practice.

The metrorrhagias do not require a detailed description of treatment. About 60 per cent are caused by cancers of the uterus, and about 12 per cent by chronic hyperplasia of the endometrium, also termed hemorrhagic myopathy or metropathy. About 15 per cent, in our series, resulted from a complication of pregnancy.

The chronic hypertrophy of the endometrium demands a more detailed discussion. The maturing follicle, for unknown reasons, may persist and continue to grow, forming a cyst the size of a walnut. The cystic follicle contains clear fluid. The walls are formed of a succulent, thick granulosa layer which rests upon a fibrous membrane and a theca interna, richly supplied with capillaries.

The uterus has a soft, loose, juicy, and bluish colored myometrium. The cervix is soft and wide. The endometrium is thickened. It represents a continuation of the premenstrual proliferative phase of the menstrual cycle. The surface may be smooth and even, or irregular, fuzzy, fungoid, or polypoid. On microscopic examination an increase of the epithelial and stromal elements is found. The glands are enlarged; many are cystic. The disparity in the glands is the most conspicuous feature. The epithelial cells are seen to be several layers thick. The stroma is markedly increased. Occasionally mitoses are noted. Necrotic degenerations of the hyperplastic mucosa are the real sources of the alarming hemorrhages seen in this disease.

About 70 per cent of endometrial hyperplasia due to a persistent ovarian follicle occur from forty-one to fifty years of age and about 25 per cent at the beginning of menstrual life, though it may occur at any age. Irregular and continued hemorrhages are the outspoken symptom. An amenorrhea of five to eight weeks, rarely longer, precedes the onset of the hemorrhage. On bimanual examination the uterus is large and soft in the climacteric metropathies, and the uterus may be hypoplastic but soft in the juvenile or pubescent metropathies; the ovary in both forms is cystic and about the size of a hen's egg.

The pubescent metropathy should be differentiated from a possible abortion and extrauterine pregnancy, the preclimacteric metropathy from a possible abortion and particularly malignant disease. Therefore, diagnostic curettage and microscopic examination of the scrapings are imperative.

The course is transient, especially if the graafian follicle ruptures or should be ruptured. Ovulation, follicle ripening, and corpus luteum formation may then again proceed normally. The endometrium undergoes resolution to normal.

The treatment of the juvenile or pubescent hemorrhages is difficult, as the cause lies in the function of the ovary and the organization of the ovum. There is a primary ovarian insufficiency plus an insufficient development of the uterus, especially of the myometrium.

The mode of living, labor and rest, diet, and exercise must be regulated. Adrenalin, parathyroid, and pituitary extracts should be given as mentioned before. Finally, the hemorrhage may be arrested by curettage, which may be repeated a second and third time. The persistent ovarian follicle may be ruptured bimanually under narcosis. In hemorrhagic diathesis x-rays may be applied to the spleen. The dose is 15 per cent of an erythema skin dose effective in the organ. If all treatment fails, the persistent corpus luteum should be resected.

The treatment of preclimacteric metropathies consists in a curettage which is curative in about 50 per cent of the cases. The scrapings must be examined histologically to exclude cancer. Recurrence of the

bleeding indicates the use of x-rays or radium.   Radiation therapy has supplanted quite a number of abdominal and vaginal corpus amputations in benign uterine hemorrhages.

SUMMARY

1. A classification of uterine hemorrhages based on the etiologic factors has been given.

2. The types of uterine hemorrhages have been grouped into (a) menorrhagias, and (b) metrorrhagias.   The menorrhagias have been subdivided into hypermenorrheas, if the menses are too profuse or last too long, and polymenorrheas, if the menses occur too soon.

3. The treatment of the conditions causing hypermenorrhea, polymenorrhea, and metrrohagia has been given.

4. Hemorrhagic metropathy has been discussed.   It is divided into the juvenile or pubescent and the climateric or menopausal hemorrhagic metropathies.   The etiology, pathology, symptomatology, and therapy have been given.   Hemorrhagic metropathy, or endometrial hyperplasia due to a persistent ovarian follicle, is the most important form of the metrorrhagias, if malignant diseases are excluded.

5. It is imperative that curettage be performed and the scrapings examined microscopically, if the cause of the bleeding cannot be determined.   Otherwise hidden malignant disease may be overlooked.

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25 EAST WASHINGTON STREET.

THE EARLY DIAGNOSIS OF CANCER

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THE question of early diagnosis of cancer is a serious one. It is strange that a disease which has been recognized for centuries, which was well known to the ancients and has been dreaded at all times, should be so insidious in its onset that we still are entirely ignorant as to its exact cause.

True it is that we do speak of certain lesions or conditions as precancerous because our experience and observation have taught us that cancer very often follows in their wake, or develops upon them. However, it does not always do so, and we do not know why it occurs in the one instance and not in the other.

Some people question whether the disease is more common now than formerly, but on the whole, competent observers and statisticians seem agreed that it occurs more frequently and is definitely on the increase.

In a disease so prevalent that we know one in every ten adults now living will die from it, and among women between the ages of forty-five and sixty-five, one in every five deaths is caused by it, we would reasonably expect that a large fund of information concerning all phases of the disease would be available, and that the earliest stages would be readily recognizable and a matter of common medical knowledge.

Let us now ask ourselves: can we make beyond a reasonable doubt a diagnosis of incipient or early cancer, based upon its gross characteristics?

In answer to such a question I imagine that each of us immediately constructs a mental picture of a lesion arising in some organ or part of the body with which we have had considerable experience.

If we recall the earliest lesions we have observed, no doubt the picture will be either a wart or a superficial ulcer with surrounding induration, or a nodule or lump of variable size.

On further thought, however, we shall probably realize that in the vast majority of instances our earliest diagnoses were accidental, were not based upon definite symptomatology, occurred in the course of treatment for a supposedly benign lesion, and were based upon routine microscopic study of tissue removed during such treatment. Moreover, in some instances the malignant nature of the lesion had not even been suspected by us and the report from the pathologist came as a great surprise.

The speed with which symptoms occur and attract the attention of the patient depends largely upon the sensitiveness of the organ in-

volved and upon the ease with which its function can be disturbed by a small lesion. For instance, a minute lesion on a vocal cord or on the tongue may attract attention promptly, whereas a neoplasm of the liver or bowel, uterus, or ovary may attain considerable size before recognizable symptoms occur.

It is evident that early clinical diagnosis of cancer in the various organs of the body is entirely dependent upon three factors: the location of the organ, the ease with which its normal functions are disturbed, and the nature of the symptoms produced by the lesion.

It is utterly hopeless based upon these three factors to hope to make an early diagnosis of internal cancer except by accident.

What are the readily recognizable factors that are common to the different varieties of the disease?

In the first place, the primary lesion is always identifiable as an overgrowth of cells that heretofore have been normal and essential to the integrity of the organ in which the disease has located. These cells, however, no longer behave as normal cells; they have acquired a tendency to rapid division and growth, and still more important, to invasion and destruction of adjoining normal tissues and replacement by the cancer cells. Even more interesting is the fact that the individual cancer cells, if broken off from the original tumor but still remaining in their host, may be carried by the lymph or blood streams to other places. These cells retain the power to grow and reproduce themselves and eventually may form a tumor which in many of its gross characteristics will resemble the original tissue from which the cell was derived.

When we, therefore, take the factors which are common to cancer in any part of the body and which may aid us in reaching an early and correct diagnosis, we have first, evidence of preceding persistent benign disease, or chronic irritation which has usually been present over a considerable period of time, the so-called precancerous lesion.

Second, we find increased size or bulk of the organ, or part involved, or a definite lump.

Third, the consistency of the tissues involved is more dense and resistant, and less elastic than normal.

Fourth, there is a definite impression, especially in the more superficial cancers, as seen on the lip, tongue, or vulva, of a piling up of cells or exudate in and under the skin or mucous membrane, forming a small localized tumor which at first does not seem to have much invasive tendency but rather soon, in fact while it is still quite small, tends to ulcerate on its exposed surface, and thereafter gradually but definitely increases in size and bulk and soon shows glandular metastasis. When an internal organ gives symptoms of sufficient degree, verified if possible by x-ray studies, to lead to surgical intervention, the disease will nearly always be found to have gone beyond

the early stage. The operation usually reveals a definite tumor, often with attachment to neighboring structures or with glandular involvement.

It is useless to talk of early diagnosis in cancer of the stomach, for instance, for it may exist without sign or symptom and be recognized only by roentgen-ray studies. On the other hand, cancer of the skin, lip, tongue, breast, vulva, penis, should be discovered promptly by the patient, and the diagnosis should be made without unnecessary delay by the physician.

Cancer of the breast presents probably the most difficult problem in this group. A single nodule with evidence of fixation should be investigated; freely movable or multiple nodules are more likely to be benign or to indicate chronic mastitis.

Carcinoma of the cervix cannot be expected to give early symptoms; there is no function to be interfered with, a watery discharge or leucorrhea is not typical and when bleeding occurs, it is in most instances due either to ulceration of the cancer tissue which is no longer able to maintain its integrity or to rupture of blood vessels from infiltration with cancer. In any case bleeding is an indication that cancer cells are in direct contact with the blood stream and with the lymphatics, and the probability of metastasis or extension from the primary site is assured.

The gross appearance of early carcinoma of the cervix is in no sense typical. In fact it is exceedingly difficult to distinguish chronic cystic and interstitial cervicitis with erosion from moderately developed or early infiltrating epidermoid carcinoma with ulceration. Erosions, in my experience, are seen in younger women, as a result of irritating discharges following abortions or full term pregnancies, or from venereal infection. Since these causative factors of erosion are less common in women over forty years of age, likewise erosions are infrequent, and when found at this time of life, they should be regarded with grave suspicion, for many of them are actually instances of incipient cancer.

The benign lesions of the cervix found in women over forty years of age are nearly always characterized by increased connective-tissue production and cyst formation and not by erosion or ulceration. Moreover, mucus predominates or forms a large part of the discharge from a benign cervical lesion and is rarely present in cancer of the cervix.

The vast majority of the cases of carcinoma of the cervix are histologically of the squamous cell or epidermoid type. A very small percentage, about 2 per cent to 3 per cent, arise from the cells of the cervical glands and are of the adenocarcinoma type.

Let us now consider the gross anatomic lesion or lesions as observed in carcinoma of the cervix. In practically every instance the cervix is larger, more bulky, nodular, and more irregular in outline than nor-

mal. There is usually a distinct asymmetry as regards the location of the external os. This increased size is due to tumor growth and invasion of the normal structures of the cervix by the neoplasm, and to some extent to increased tissue production and lymphocytic reaction as a defensive or protective measure.

Our experience leads us to believe that carcinoma of the cervix seldom begins as a superficial ulcer and only rarely ulcerates early in the disease. The exceptions are the cases of papillary, fungating, cauliflower carcinoma, which form huge, friable, spongy masses of newgrowth on the surface of the cervix and for a long time show only slight tendency to invade the normal tissues. There are also a very small number of slow-growing, prickle-cell, squamous, epidermoid carcinomas, which invade slowly, ulcerate early, remain comparatively superficial for a long time, closely resemble a chancre, and are very resistant to ray therapy.

Early diagnosis of cancer, I believe, in the light of our present knowledge, is largely a speculative matter, and yet its early recognition is important, for, if curable at all, it is only in its initial state.

According to Maud Slye "two factors are necessary to produce cancer, one, an inherited susceptibility to the disease, and the other, irritation of the right kind and in the right degree applied to the cancer-susceptible tissues."

In the case of a suspicious nodule in the breast we shall do well to remember Bloodgood's statement that "the warnings of cancer are not different from those of the local lesions which are not cancer" and that early benign but precancerous breast tumors "can be differentiated only at the exploratory operation, and that the final decision rests upon the microscopic study."

Most of us feel that cancer seldom if ever begins as cancer but is preceded always by certain well-recognized precancerous conditions in various portions of the body, such as syphilis of the tongue, leucoplakia of the vulva, chronic endocervicitis and laceration of the cervix, chronic mastitis, warts, birthmarks, moles, burns, scars, etc., which if present or known to have been present at the site now occupied by the neoplasm, increase the probability of its being cancer.

As a final conclusion, however, we can say that biopsy with microscopic examination of the tissue by a competent pathologist is essential in the early stage of growth of the lesion for exact diagnosis of cancer. It is, of course, possible that serologic tests may eventually open the door to early diagnosis without biopsy but at present there is none that is reliable. As W. Sampson Handley has well said, "The detection of early cancer is not a one-man job. It may require the cooperation of the patient, the family doctor, the specialist, and the pathologist. It may be impossible even then, in some cases."

121 EAST SIXTIETH STREET.

PREMATURE SEPARATION OF THE NORMALLY IMPLANTED PLACENTA WITH SPECIAL REFERENCE TO THE KIDNEY IN THESE CASES

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PURPOSE.—It is not the purpose of this paper to reconsider the question of premature separation of the normally implanted placenta from the standpoints of multiple etiology, pathology, symptomatology, classification for obstetric treatment, obstetric treatment itself and results or bibliography. Williams,[21, 22] Holmes,[12, 13] Polak,[17] Willson,[23] Portes,[18] Phaneuf,[19] and others have well covered these aspects of the condition. Goethals, of the Boston Lying-In staff, working independently has just now done the same with the series from which the bulk of our material is taken.

Our purpose at the start of this study was to link up the group of these cases in which the kidney is involved with our previous work on the "toxemic-chronic nephritic" group. We sought to clear up in our own minds certain questions regarding the kidney which presented themselves to us as we debated treatment in a given case. We felt that though much had been accomplished along lines of obstetric treatment by our predecessors, much room was left for a better understanding of the treatment in relation to the kidney. We felt that if we could obtain this understanding by study, we could lower our mortality rate. We were particularly impressed by the group of patients with separated placentas—clinically distinct from anuric eclamptic, patients who showed marked anuria. We were totally at a loss to explain the urine and blood chemistry findings observed over a period of days in some of these cases by the pathology implied by the terms chronic nephritis and acute nephritis. We hoped that a study of our material would throw some light on these and other puzzling questions.

Literature.—From the voluminous literature on "separated placenta" we wish to consider only those contributions which bear on the subject under discussion.

The association between nephritis and premature separation of the placenta, according to Essen-Möller[7, 8] was first pointed out by Chantrenil in 1881. Winter,[25] in 1885, presented three cases and noted the occasional presence of nephritis with the condition. Secondarily, he advocated endometritis as an etiologic factor. His observations were corroborated by Hennig,[10] Weiss,[24] Lehman,[14] and Hofmeier.[11] Holmes,[12] in 1901, was not impressed by the presence of albuminuria in these cases. He stated that "kidney changes" were noted only 20 times in 200 cases, and he believed that endometritis was the principal factor.

In 1903 Muus[15] and in 1905 Paul Bar[2, 3] suggested that the same intoxication which caused eclampsia was also the cause of accidental hemorrhage. Bar and Kervily[3] later reported the death of a patient from premature separation of the placenta. Liver changes similar to those in eclampsia were found. These are the first observations in the literature relating to premature separation of the placenta, toxemia of pregnancy, and eclampsia.

It remained, however, for Essen-Möller, in 1913, to emphasize the important connection between toxemia of pregnancy, eclampsia, and premature separation of the placenta. He says, "And last of all it is striking how often the general state of the patient in a case of accidental bleeding resembles an intense intoxication, as we see it in eclampsia. But the other reasons I have mentioned speak very much in favor of the supposition that accidental hemorrhage and eclampsia are genetically connected. The increased blood pressure, which is so common in eclampsia, may also be of some importance for the separation of the placenta. The albuminuria would thus only be a symptom of the general intoxication which in one case causes eclampsia, in another case accidental hemorrhage, in a third perhaps both these diseases. But this does not of course give any explanation as to the origin of the accidental hemorrhage. The problem will then form a part of the great problem of eclampsia or of the toxemia of pregnancy. But the fact that the two groups of symptoms which we formerly took to be indicative of two different diseases, can be looked upon from a common point of view, seems to be an advantage."

Williams believes that from observations of the relation of premature separation to eclampsia, there must be some connection between it and the toxemic process. However, he is unwilling to concede that the concomitant toxemia is of the pre-eclamptic variety but "is of a special type concerning whose causation we are at present ignorant."

DeLee[5] mentions the frequent association of toxemia of pregnancy and nephritis and the premature separation condition. Ahlström,[1] Shaw,[20] and Portes in particular, believe toxemia of pregnancy is the underlying factor in all except the occasional unexplained case.

Particular reference to renal involvement or renal disease in the literature is not made except in the earlier writings already described or in an occasional general statement. Williams[21] noted albuminuria in 11 out of 29 cases, Gaston,[9] 30 out of 70; Bar and Kervily, 38 out of 58; Dorman,[6] 82 out of 158, and Essen-Möller, 11 out of 29 cases. However, later authors note a much higher incidence of albuminuria in their collected reports. Willson (1922), in reviewing 69 cases, notes that albuminuria was present in 86.2 per cent of cases in which the urine was noted. He was able to find only 9 instances in which the blood pressure readings were recorded. The average systolic pressure was 182 mg. Hg. Clinical or pathologic evidence of toxemia was found in 87.7 per cent of 57 cases. Eight and six-tenths per cent of the patients showed eclamptic convulsions. Strikingly similar are the findings of Portes in 1923. In a report of 73 cases he found clinical evidences of toxemia of pregnancy in 91.3 per cent, albuminuria in 88.5 per cent, and eclamptic convulsions in 8.3 per cent.

Mention of complications which may follow premature separation of the placenta is restricted to the description of postpartum hemorrhage. We have been unable to find suggestions by any author that complete or partial suppression of urine associated with premature separation of the placenta, either with or without eclampsia, is an important or dangerous complication. In a number of reported cases in which the urinary output is described, there is, however, a definite urinary suppression. Couvelaire[4] in a report of two cases (1912) mentions a para v, thirty-one years old, who was transported to the operating room in extremis. A Porro-cesarean section was performed. This patient had a partial suppression, certainly during the first two days postoperative, and died on the third day. His second

case, a primipara, eclamptic, with premature separation of the placenta delivered normally, and had thirty-four hours with absolute anuria for two days postpartum. A bilateral decapsulation of the kidneys was performed, but the patient died thirty-four hours after delivery. On autopsy, edema of the perirenal tissue was found. The kidneys showed only congestive changes consistent with capillary dilatation. The convoluted tubules were occupied with cellular detritis and fine granular cylinders.

Oldfield and Hann[16] report a more typical case. Their patient was a primipara, thirty-eight years old. with signs and symptoms of premature separation of the placenta. At the time of operation only 1.5 ounces of blood-stained urine was obtained, "loaded with albumin." Complete urinary suppression followed for thirty-six hours and partial suppression for three or four days, after which the urinary output increased, and the patient made an uneventful convalescence and was albumin-free on the seventeenth day. These authors are the first and only ones, as far as we have found, to note that urea excretion was diminished and to describe the urinary sediment.

Material.—The original material on which this study is based consists of approximately 165 cases indexed as "separated placenta." All but 13 of these occurred in the last ten years of the Boston Lying-In Service. The others were seen by me (F. S. K.) in my private practice and are included because most of the patients have been under observation three years or more, a point of great importance. These 165 cases briefed and studied were immediately reduced in number to 60. The other patients had definitely separated, low-attached placentas, or were definitely traumatic, or gave insufficient recorded data to make them of any value to us. The remaining 60 cases were all associated with what we have called the "toxemic-chronic nephritic group." Intensive study of these 60 cases led us to omit 31 more. The reason for this reduction was that though they belonged in the group, we saw that the data were insufficient to advance our ideas. Of the remaining 29 cases, each, for one reason or another, seemed to us to possess more merit for study than the usual reported case.

The Groups.—It is impossible in a paper of this length to enter into the detail of these 29 cases. If we indicate why we venture to state that they possessed more than average merit for study; if we detail the single case which first brought to a head our interest in this subject; if we then offer our opinions based on this work, and draw such conclusions as we may, we shall have accomplished all that our time permits. Nor are we disposed at present to publish the cases as they stand in protocol, for the reason that in the next few years opportunity for interval study in most of the surviving cases will have occurred. We therefore offer our effort to you in a tentative and preliminary form.

Our best method of indicating briefly why we think these cases are more valuable than average seems to be to group them under certain headings. We studied them in this way because each group contained the suggestion of a possible answer to one or more of our questions.

Please bear in mind that we are not yet concerned with statistics and figures, that some of the cases fell into more than one group, and that, therefore, the addition of the group figures which we present will not and should not equal the 29 patients from whom the material came.

Group 1.—Five cases not accompanied by eclampsia in which complete anuria was present in 2, and marked incomplete anuria in the other 3. Of these patients 3 died and 2 recovered.

Group 2.—Five cases associated with eclampsia. We would emphasize one feature common to these 5; namely, that each had had her convulsion prior to placental separation.

Group 3.—Eleven cases in which satisfactory and reasonably complete blood chemistry, blood pressure, and urinary data were recorded.

Group 4.—Eight cases. Each patient had been in hospital for treatment of toxemia of pregnancy with elevated blood pressure, albumin in urine, and other symptoms, and had cleared so completely that she was either just home, or on the eve of discharge, when separation occurred.

Group 5.—Twelve cases in which we have partial adequate interval study, or a definite history of marked toxemia in more than one pregnancy.

Besides studying these groups, we think we have made three other observations in this condition. We know or suspect these have been previously made, and we insert them here simply as confirming the observations of others. (1) These patients have high white counts (in so far as our limited data go) and, since high white counts may result from throwing foreign protein into the blood stream, we have a certain amount of added theoretical evidence that this group of separated placentas is caused by a protein toxic agent. (2) Certain of these patients show increased bleeding and coagulation time. (3) As these patients entered the hospital, inspection, as a rule, classified them into one of two types. The first of these is the nephritic type; generally characterized, we think, by relatively moderate parity when seen. The second type, older in years, stringy, worn, many para, looking like the more usual hospital patient with placenta previa. We think that it is in this type that endometritis may be the main etiologic factor; whereas, in the other group we feel convinced that the same etiologic factor as in toxemia plays the major rôle in the premature separation.

Having as briefly as possible outlined the cases in groups, we shall cite in detail the history of a patient who increased our interest in the pathology and treatment of toxic premature separation. She led us to an intensive study of the literature and to a feeling that we could not be content with it. Especially were we impressed by the slight amount of available information, to say nothing of the marked disagreement of authorities, concerning kidney pathology in these cases. We sought further information from the genitourinary surgeon, from the internist, and lastly by good chance from a well-known

student of the physiology of the kidney, and we found only a little suggestion of help from them. It must be said that what actually took place in the kidneys of the patient about to be described is not yet known. Autopsies as we have seen them, and those cited by Willson, do not help. They are few in number and by no means meet the requirements of the clinical picture. We suspect, as with our own, that the ones he reports were done on patients dying relatively soon after delivery. The single autopsy finding, Couvelaire's second case in which at autopsy, "edema of the perirenal tissue was found, the kidneys showed only congestive changes consistent with capillary dilatation; the convoluted tubules were occupied with cellular detritis and fine granular cylinders," corresponds to the clinical picture in those anuric patients who recover.

THE CASE

M. B., para i, forty years old, had scarlet fever when four years old; also tonsillitis when a child. She first came to the prenatal clinic Jan. 21, 1927. Last period was Sept. 1, 1926. Expected date of confinement June 8, 1927. On Oct. 1, 1926 there was slight antepartum bleeding.

On this first prenatal visit, the patient had a systolic pressure of 160. No albumin. According to our system she was referred to the hospital for study. Hemoglobin, 30 per cent; red count, 3,516,000. Blood chemistry was within normal limits (see Table II). Eyegrounds negative. Pthalin 35 per cent. One hour renal test was normal.

TABLE I

HOURS	OUTPUT	INTAKE		B. P.
1st 24	4 drops	Subpect. Sal.	1500 c.c.	Low 100/70
		25% glu. intra.	300 c.c.	
		Subpect. Sal.	1600 c.c.	High
		Transfusion	500 c.c.	120/80
			3900 c.c.	
		By mouth and rectum up to	6360 c.c.	
2nd 24	45 c.c.	Subpect. Sal.	1500 c.c.	Low 125/65
		25% glu. intra.	250 c.c.	
			1750 c.c.	High
		By mouth and rectum up to	6270 c.c.	170/80
		Sal.	3000 c.c.	
		Glu. intra.	250 c.c.	
3rd 24	135 c.c.	By mouth and rectum up to	3860 c.c.	Low 150/85 High 200/120
4th 24	267 c.c.	All by mouth	2610 c.c.	Low 155/85 High 200/120
5th 24	217 c.c.	Subpect sal.	1500 c.c.	Low 160/85
		By mouth and rectum up to	3460 c.c.	High 180/95
	Total 634 c.c.	Total	22560 c.c.	

During eight days in the hospital her blood pressure dropped to 120/80. She was discharged to the toxemic clinic. She reported there four days later, and from February 11 to March 8 her blood pressure ran from 154 high to 136 low, and no albumin.

At 3:00 P.M. on March 27, 1927, she vomited and lost one ounce of fluid blood from the vagina. She felt giddy and faint, had blurred vision, but no loss of consciousness, headache, or abdominal pain; fetal movement ceased. On entrance to hospital blood pressure was 160/100, pulse 120, temperature 98.2°; uterus was board-like and tender; fetal parts could not be mapped out, and fetal heart could not be heard; there was slight red vaginal staining. No rectal or vaginal examinations were made. Reflexes were active; she had moderate edema of extremities, and slight edema of face and retina. Urine at this time was 4 c.c. in amount, cloudy red, with trace of albumin, occasional red blood cells, hyaline, and granular casts.

To recapitulate: February 7, 1927, a patient is discharged from hospital with normal blood pressure and urine, negative blood chemistry, and negative kidney tests. She is followed as carefully as possible in the special toxemia clinic. Fifty days later she appears, an undoubted case of toxic separation, in excellent condition for section, and with a good prognosis in comparison to that of a similar patient bled out. Cesarean section, confirmation of the diagnosis by the appearance of the uterus, separated placenta, dead baby, and free blood and clots. Uterus acted well and is left in, little postpartum bleeding; pulse, and pulse pressure satisfactory.

By this time we have learned that hemorrhage is not the only thing that may kill a patient with separated placenta, even in the noneclamptic group, and that speedy surgery applied early and ample transfusion do not always save such patients. Accordingly, the patient is catheterized every eight hours, has four-hour blood pressure readings, and these observations are checked up against our treatment. Table I shows results by twenty-four hour periods.

From the fifth day on, the output steadily rose to a maximum of 112 ounces on the ninth day when that side of the matter was considered closed. During these nine days the albumin dropped to the slightest possible trace, then cleared up except occasional S. P. T., apparently from pus in the urine. The urine became pale, and casts and cells disappeared in five days. The blood pressure dropped to 160, and ultimately to 120.

The use of the 25 per cent glucose intravenously three times in the effort to open the kidney was checked up by blood-sugar tests, done as part of the routine blood examinations. These findings are accordingly shown. (Table II.)

The patient, after a stormy convalescence with low grade sepsis in wound and uterus, was ultimately discharged obstetrically well to the Boston State Hospital for the Insane, where she had previous admissions.

These tables taken from the case detailed above, and more or less like the other anuric cases we have studied, illustrate several points.

TABLE II

DATE	N. P. N.	BLOOD UREA NITROGEN	BLOOD URIC ACID	BLOOD SUGAR	BLOOD CHLORIDE (CHECKED HIGH, LOW)
2/ 2/27 Day of 1st ad.	26	8.5	31	74	
3/28/27 Day after del.	59	31.9	6.7	143	554 543
3/29/27	64	38	7.3	111	566 566
3/30/27	86	50	8.8	143	554 554
3/31/27	67	32.7	10.7	117	531 531
4/ 1/27	100	66.1	11.4	*166*	508 496
4/ 2/27	110	66	10	122	507 496
4/ 3/27	*120*	70	10	126	508 531
4/ 8/27	60	41	8	100	554 543
4/15/27	33	16.9	4	52	519 508
6/ 1/27	18	7.2	2.7	81	514 508
6/11/27	27	9.3	2.9	100	496 496

Mg. per
100 c.c.
Whole Blood

These are (1) the progressive slow recovery of the kidney when it begins to secrete; (2) the tremendous intake of fluids we use in the effort to force the kidney to secrete; (3) at first the progressively increasing blood pressure and later its high stabilization between high point and low point, accompanied by the progressive urinary output; (4) the use of concentrated glucose intravenously to produce hyperglycemia, in an effort to obtain diuresis. This is checked by the daily blood-sugar findings as illustrated in Table II by the figure 166, which represents the culmination of three days of intravenous glucose therapeutically. At this figure or somewhat below, sugar appears in the urine. (5) It illustrates that the kidney must recover its ability to secrete nonnitrogenous material including water, first because it shows the maximum of nitrogen-retention on the seventh day of the puerperium while at this time large amounts of urine of high specific gravity are already being excreted. This is as one would expect from the long time necessary to produce uremic death and uremic blood findings experimentally. One patient in our anuric series died on the sixth day with uremic blood findings and in typical uremic coma with uremic twitchings, though she was not an eclamptic on entrance.

Furthermore, Table II illustrates a patient who comes to us with a history, of an age, with appearance, with blood pressure—all suggestive of chronic nephritis, but whose blood chemistry, eyegrounds, and reaction to treatment demonstrate that she is not a chronic nephritic

in the accepted sense of the term. She then passes through an almost complete kidney shut-down and during this time shows urinary findings and blood-chemistry findings approaching those of a woman about to die of uremia. Yet two months later she emerges with the same normal blood chemistry, urine, and blood pressure that she had in the beginning. If we attempt to place the rôle of chronic nephritis in these cases, we are in no position to do so accurately with our present knowledge. The appearance of albumin in the urine, elevated blood pressure, etc., in more than one pregnancy does not permit of this diagnosis on the one hand, and on the other, retained nitrogen products in the blood does not mean simply chronic nephritis, but means a decompensated nephritis, since upwards of two-thirds of the kidney tissue must be out of commission before sufficient nitrogenous products are piled up to be demonstrable in the blood. We have in this series data of four cases which lead us to believe that they are at present, and presumably were chronic nephritic patients at the time of separation, but we are not yet sure. We have a feeling that not a few will turn out to be chronic nephritic patients, but this feeling is based more on the inspection of the patient than on anything we are yet able to demonstrate. When we make this statement, we are sensible of the observations made in other toxemic clinics, particularly in Baltimore and in New York, as well as in our own, on the development or at least on the discovery of chronic nephritis subsequent to various forms of toxemia of pregnancy. As yet, these are not convincing to us in anything like the proportion of cases so described. We feel that only by many years of observations of many of these patients, or better by the development of some more delicate test of kidney insufficiency than has yet been offered, can the true rôle of chronic nephritis be known in separated placentas.

CONCLUSIONS

1. We believe that the major result of this study has been to show us more tangibly than before how we should approach and follow each case of so-called toxic separated placenta.

2. We believe that anuria in separated placenta is a complication to be feared and to be combated as we have outlined, from the first moment that the patient is seen, with forced fluids by mouth and rectum and under skin, glucose 25 per cent in vein, and usually transfusion, fluids up to from 6000 to 7000 c.c., checked up by daily blood chemistry, by blood pressure and by eight-hour interval catheterizations with urine analysis.

3. We believe in this connection that the preservation of a relatively high blood pressure is an important part of the treatment and that it represents a protective mechanism against the anuria.

4. We believe that in the eclamptic group of separated placentas, dropping pressure to control convulsions should be exercised with great caution lest the patient be aided to become anuric.

5. We believe that on the chances the etiologic factor in toxemia of pregnancy and toxic separation of the placenta is sometimes, at least, the same. We base this belief on the fact that we have been able to collect 8 cases of definite toxemia without convulsions (nonnephritic as far as we can say at present) that separated just as the signs and symptoms of toxemia had abated.

6. We believe from this group that extreme supervision should be exercised after a toxemic patient has improved to the point of discharge, and that she should be warned to remain quiet and report if she has the slightest bleeding or abdominal pain.

7. We believe that the explanation of what actually takes place in the kidney in these cases, whatever its pathologic background, must be sought in further study of the physiologic exchange in the kidneys, rather than along lines of autopsy pathology.

8. We believe that decapsulation of the kidney has no place in these cases.

9. We believe that every effort should be made by every obstetrician and clinic to link up these cases with the "toxemia-chronic nephritic" group, and study them with careful tests and gather all available data, over the longest possible period. This so that as the scientific laboratory comes forward with more light on the etiology or etiologies of these conditions, the clinical laboratory will not be behind in case groupings to fit.

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THE PROGNOSIS IN ACUTE AND CHRONIC NEPHRITIS OF PREGNANCY FROM THE STANDPOINT OF CLASSIFICATION

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THE physician is frequently consulted concerning prognosis in cases of pregnancy or on the advisability of future pregnancy if a woman has symptoms of nephritis or has had nephritis or toxemia in a previous pregnancy. The opinion prevails that a woman with a history of nephritis should be advised against future pregnancies, and yet careful observation of such a case may reveal that the function of the kidney is more than sufficient to carry the patient through pregnancy under careful supervision. Conversely, if renal injury is serious, the physician should advise against future pregnancy, until a careful rechecking of conditions shows that renal function is adequate. The modern conception of nephritis is that it is a local manifestation of a diffuse disease affecting the entire vascular system. The disease varies in degree and extent, depending on the potency of the toxin attacking the vascular system and on the organ or organs most severely affected. Study of the nail-fold capillaries in eclampsia reveals changes similar to those in acute nephritis, such as areas of contraction and dilatation of the capillary loops. Changes in the retinal vessels are further demonstration of the widespread vascular character of the disease.

The vascular involvement may be generalized and without apparent renal injury. The vessels may return to normal after confinement, or so-called benign hypertension may follow, or the injury may be the beginning of progressive arteriosclerosis finally terminating in malignant hypertension. In such widespread vascular injury, the kidneys often bear the brunt of the attack and the capillary tufts of the glomeruli can be so seriously injured that complete recovery is not possible and renal function is chronically impaired. Similar injury to the liver may pass unnoticed because of the wide margin of safety which must be overcome before symptoms of hepatic insufficiency are manifest, and because of the liver's strong power of regeneration.

Clinical evidence indicates that the kidneys are affected in most cases of preeclamptic or eclamptic toxemia. Volhard emphasizes the resemblance between true eclampsia and the convulsive form of uremia. One of us (Mussey) has noted the close similarity of preeclamptic toxemia and eclampsia on the one hand and acute glomerulonephritis on the other.

In a study of 100 cases of renal injury resulting from pregnancy, Rockwood, Keith, and Mussey found that the nephropathy of pregnancy could be grouped according to Volhard and Fahr's classification of nephritis as follows:

        A. Pyelitis and pyelonephritis
        B. Hypertension and nephritis
            1. Acute nephritis
                a. Acute glomerulonephritis
                b. Acute nephrosis
            2. Chronic nephritis
                a. Chronic diffuse glomerulonephritis
                b. Chronic nephrosis
            3. Sclerosis (vascular lesion)
                a. Benign hypertension
                b. Malignant hypertension

The pyelitis and pyelonephritis complicating pregnancy are relatively common; they will not be discussed here.

HYPERTENSION AND NEPHRITIS

Important findings in acute glomerulonephritis are hypertension, edema, and albuminuria usually with erythrocytes in the urine and lowered urine output. Although this disease is described as inflammation confined chiefly to the glomeruli, it is usually associated with more or less evidence of tubular injury. Nephrosis is described as a degenerative lesion of the tubules. Its chief features are marked edema with lowered urine output, albuminuria, usually casts, but few, if any, erythrocytes and no hypertension. In acute nephritis of pregnancy, these two lesions are frequently associated so that Fahr and Baer define them by the term glomerulonephrosis.

The diagnosis of acute nephritis and acute nephrosis in pregnancy rests chiefly on clinical data. Rarely is there phenolsulphonephthalein retention, or increase in the nonprotein nitrogen in the blood. While laboratory examinations, aside from tests for albuminuria, are not of great value in the acute nephropathy of pregnancy, they are often of distinct value in the differential diagnosis and prognosis of chronic nephropathy. Several types of chronic nephritis may develop as a sequence of renal injury which has not resolved following confinement. It is evident that the degree of interference with renal function varies with the type of lesion and the extent of involvement. We found that in most cases classified as acute nephritis the patients recovered completely, although in some cases the disease progressed to the chronic stage. In cases diagnosed focal nephritis, nephrosis, or benign hypertension, the prognosis was favorable, while in those classified chronic glomerulonephritis, malignant hypertension, and chronic (diffuse) nephritis, the prognosis was poor.

This study demonstrated that the classification of nephropathy resulting from pregnancy is of value in prognosis. By applying this classification in cases of pregnant women with symptoms of nephritis and of women in the childbearing age with nephritis, in combination with tests of renal function, we have been able to determine the prognosis in certain cases more accurately.

These women are given a careful general physical examination, and previous illnesses, particularly those complicated by nephritis, are noted. If there is evidence of edema or hypertension, if albuminuria is present, or if there is a history of previous renal trouble, further tests are made. Increase of urea or nonprotein nitrogen in the blood, or the prolonged retention of phenolsulphonephthalein is evidence of lowered renal function. If function is seriously curtailed, the specific gravity of the urine is relatively fixed. While in normal persons on a dry diet, the urine will be concentrated and the specific gravity should rise to 1.025 or more, conversely, following the drinking of a quantity of water in a short space of time, the specific gravity of the urine should be distinctly lowered.

Conclusions may be drawn more accurately if the doubtful case is examined in hospital. The patient is kept in bed on a prescribed diet and fluid intake for several days during which time blood-pressure readings are recorded and the ocular fundi examined. The output of urine is measured, the phenolsulphonephthalein test is made, and the nonprotein nitrogen in the blood is determined. Two days are set aside for determining the upper and lower limits of the specific gravity of the urine. For the concentration test, the patient is given food for a day consisting of 20 per cent solids only; no fluids are given. The urine is collected at three-hour intervals. The specific gravity should reach 1.025. In the water test, 1500 c.c. (seven and a half glasses) of water is given on an empty stomach between 8:00 and 8:30 A.M. The urine is collected every half hour for the next four hours. The normal output varies between 1200 and 1800 c.c. and the specific gravity should be as low as 1.003.

ILLUSTRATIVE CASES

CASE 1: NEPHROSIS.—A woman, aged twenty-seven, was pregnant for the second time. There had been no untoward symptoms during the first pregnancy. She was under observation during the last five months of the second pregnancy; slight edema appeared in the ninth month and the urine contained albumin, graded 4, but there was no hypertension. The hemoglobin was 60 per cent.

After confinement the edema and albumin disappeared. Since then a third pregnancy has been normal.

CASE 2: NEPHROSIS.—The patient,* a primipara, aged twenty-one, was first seen in labor. There was edema, graded 3, and albuminuria, graded 4, but the urine contained no casts nor red blood cells; the blood urea was normal and there was

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*Case 3 in: Rockwood, Reed, Mussey, R. D., and Keith, N. M.: Surg., Gynec. and Obst., 1926, xlii, 342-350.

no elevation of blood pressure. The patient was delivered of a healthy child. Since that time the patient has been delivered three times without recurrence of the renal symptoms.

CASE 3: ACUTE GLOMERULONEPHRITIS.—A woman, a primipara, aged twenty-five, had slight edema and albuminuria at fifth month, which disappeared under treatment but reappeared ten days before she came under our care at the thirty-fifth week of gestation. The systolic blood pressure was 168, the diastolic 100. There was edema, graded 3, and the urine contained albumin and casts. The ocular fundi were normal. In the next six days, in spite of active treatment, all symptoms increased in severity. Five days later, the systolic blood pressure was 182 and the diastolic 108. Lobulated detached areas appeared in both retinas. Following confinement, the blood pressure dropped, the edema of the retina disappeared, the retinas became reattached and vision became normal.

A second pregnancy was not attended by any recurrence of the toxemia or ocular symptoms, and the blood pressure, renal function, and the blood urea were normal. A few old pigment changes were visible in the ocular fundi.

CASE 4: ACUTE GLOMERULONEPHRITIS.—The patient, a primipara, aged twenty-one, was examined in the thirty-fourth week of pregnancy. She had been troubled by headache and blurred vision, and progressive edema had developed in the last ten days. The systolic blood pressure was 176, the diastolic 96. There was edema, graded 3, and the urine contained albumin and casts, graded 2. The ocular fundi showed contraction of the arteries, moderate edema, and detachment of the retina. Labor was induced, and the uterus was emptied the following day. Two days later the ocular condition was much more acute, as there was generalized edema with large lobulated detached areas. Following this, improvement was rapid.

Subsequent examinations have shown normal blood pressure, no albuminuria and normal renal function. The eyes have returned to normal. If this patient is kept under close observation, pregnancy should be carried through successfully.

CASE 5: CHRONIC FOCAL NEPHRITIS (?).—A woman, aged twenty, with a child aged five months came to the clinic September 6, 1922. As a child she had had scarlet fever without complications. In 1917 and 1918 she was ill and Bright's disease was diagnosed. She was well until anuria appeared two days prior to labor in April, 1922. No anesthetic was given on account of the renal condition. There were no convulsions.

At examination, the patient complained of tiring easily. The patient weighed 114; there was no edema, and only a small amount of albumin in the urine; the systolic blood pressure was 105, the diastolic 56; the blood urea was 23 mg.; the phenolsulphonephthalein test gave a return of 55 per cent; the eye grounds were normal; the tonsils were infected.

A tentative diagnosis of chronic focal nephritis was made. Tonsillectomy was performed. Two years later the patient reported that she had no trouble during a subsequent pregnancy, and since then cholecystectomy and an operation for laceration had been performed.

CASE 6: RECURRING (?) TOXEMIA OF PREGNANCY.—The patient, a primipara, aged thirty, came to the clinic January 23, 1923. She had had one miscarriage at eight weeks, a year and a half before. She was six months pregnant and had vomited for the first three months. The systolic blood pressure was 206, the diastolic 144; the pulse was 104; there were edema, graded 3, retinal changes of a nephritic type, and albuminuria, graded 4; the phenolsulphonephthalein return was 35 per cent; later it was 65 per cent; blood urea was 22 mg. She was delivered of a macerated fetus four days after her arrival at the clinic.

When she was dismissed, February 24, the systolic blood pressure was 150, the diastolic 95, the urine contained albumin, the phenolsulphonephthalein return was

55 per cent, blood urea was 18 mg., and there was no edema. By water test, the specific gravity of the urine was 1.002; by concentration test, it was 1.028. In 1926 the urine contained no albumin, and her blood pressure was normal. It was believed that this was a case of cured acute glomerulonephritis. However, in April, 1926, the patient wrote that when six months pregnant she caught cold, albumin appeared in the urine, and she was delivered of a dead fetus. Following this the albuminuria disappeared. This may be a case of the recurrent toxemia of pregnancy described by Kellogg.

CASE 7: SEVERE ACUTE GLOMERULONEPHRITIS WITH HEALING.—The patient, a primipara, aged thirty, consulted us July 29, 1924. The systolic blood pressure was 188, the diastolic 144; there was edema, graded 3, albuminuria, graded 3, and the blood urea was 71 mg. Six days later there was spontaneous delivery of a seven-months' living fetus. Under treatment the patient improved steadily and has regained her normal strength.

July 6, 1925, the systolic blood pressure varied between 134 and 118, and the diastolic between 90 and 70; the phenolsulphonephthalein return was 60 per cent and the blood urea was 17 mg. There was at times a trace of albumin in the urine. By water test, specific gravity was 1.002 with an output of 1345 c.c.; by concentration test, 1.030. Healed glomerulonephritis was diagnosed. The patient was advised to avoid pregnancy for one or two years and to have the renal function reinvestigated.

CASE 8: MILD CHRONIC NEPHRITIS.—A woman, aged thirty-three, who was pregnant for the fourth time, had been advised to have a three months' pregnancy interrupted on account of nephritis. She consulted us January 26, 1925. Six and a half years previously there had been miscarriage of a dead fetus at five months following "flu." Two and a half years later labor was induced at seven months on account of hypertension with nephritis. The child is living and in good health. A year later there was spontaneous birth of a six months' dead fetus. The patient had had excellent antenatal care, there was no albumin in the urine, and she felt well until twelve hours before labor when there was a sudden disturbance of vision which lasted for two weeks. The systolic blood pressure had ranged between 150 and 158 since the second pregnancy, but the patient felt well.

At examination, the systolic blood pressure was 164 and the diastolic 120; the urine contained albumin, graded 2, but no casts; there were few hypertensive changes in the retinal arteries. After ten days in the hospital, the systolic blood pressure was 138 and the diastolic 90; there was no albumin in the urine, the phenolsulphonephthalein return was 70 per cent; the blood urea was 14 mg., and the hemoglobin was 85 per cent. By water test, the specific gravity of the urine was 1.002; by concentration test, 1.028.

The patient was advised to continue the pregnancy. Under careful observation and on a strict diet, she was spontaneously delivered of a living child at the eighth month, following spontaneous rupture of the membranes. There had been no more albuminuria, and the systolic blood pressure did not exceed 150 after the period in hospital at the third month.

CASE 9: MILD CHRONIC NEPHRITIS.—The patient came to the Mayo Clinic in May, 1926, seven months pregnant. There had been two previous stillbirths, one in 1915, at term, when craniotomy was performed; another in 1924, when a macerated seven-and-a-half-months' fetus was delivered. During the second pregnancy she was under our observation at the fourth and seventh months. At the fourth month the systolic blood pressure was 150 and the diastolic 90; there was a trace of albumin in the urine, and no edema. The patient did not follow instructions, and when she returned at the seventh month, the systolic blood

pressure was 186 and diastolic 124; there was albuminuria, graded 3, edema, graded 2, and the blood urea was 18 mg. The patient again failed to follow instructions and two weeks later had premature labor and delivered a dead fetus.

At examination, May, 1926, the systolic blood pressure was 140 and the diastolic 80; there was no albumin in the urine. This time the patient followed instructions and was delivered at the eighth month of a living child. Two months later the systolic blood pressure was 137 and diastolic 80; the urine contained albumin, graded 1; the phenolsulphonephthalein return was 55 per cent, and the blood urea was 17 mg. We believe that in this case low-grade chronic nephritis is present, but that renal function is adequate to withstand pregnancy under carefully controlled conditions.

CASE 10: CHRONIC NEPHRITIS WITH HYPERTENSION.—The patient, aged thirty-three, mother of three children, consulted us January 19, 1925. The last pregnancy had been terminated at seven months (October, 1924) on account of marked albuminuria, casts, a systolic blood pressure of 230, and impending eclampsia. At this time, the systolic blood pressure was 200 and the diastolic 122; the hemoglobin was 68 per cent; the urine contained albumin, graded 4, casts, graded 1, and red blood cells, graded 2, and had a specific gravity of 1.021; the phenolsulphonephthalein return was 60 per cent, and the blood urea was 22 mg. The ocular fundi showed mild retinal arteriosclerosis of the hypertensive type. The patient was in the hospital for eight days, after which the systolic blood pressure was 158 and the diastolic 128; there was a trace of albumin and an occasional pus cell in the urine. By water test, the specific gravity of the urine was 1.004, and the output 1565 c.c. No concentration test was made.

Pregnancy was advised against until further observations could be made. April, 1926, the systolic blood pressure was 180; there was no albumin in the urine, and the patient was able to do most of her work.

In this case subacute glomerulonephritis had probably developed into mild chronic nephritis with hypertension.

CASE 11: SUBACUTE DIFFUSE NEPHRITIS.—A woman, aged thirty-five, who had been twice pregnant, consulted us January 17, 1925. In May, 1924, when she was six months pregnant, albuminuria had appeared; later there had been hypertension and edema, but no headache or eye trouble. Labor occurred at eight months; it was prolonged and a stillborn babe was delivered.

Eight months later the patient returned for examination to determine whether it was safe for her to become pregnant.

The systolic blood pressure was 220, the diastolic 130; there was albuminuria, graded 2, and the phenolsulphonephthalein return was 50 per cent. The eye grounds showed considerable retinal arteriosclerosis. After the patient had been in the hospital for nine days, the systolic blood pressure was reduced to 168, the diastolic to 110; the urine contained only a trace of albumin and a few red blood cells. By water test, the specific gravity of the urine was 1.003 and the output 1200 c.c.; by concentration test, the specific gravity was 1.025.

Subacute diffuse nephritis of pregnancy was diagnosed. The patient was advised against further pregnancy until defective teeth had been removed and until she could be reexamined at the end of a year. We learned that she became pregnant shortly after returning home. In August, 1925, she wrote that she was "fighting albumin in the urine" and expected confinement in November. The outcome in this case is not known. The symptoms of nephritis had probably recurred during pregnancy because the patient had not waited until they had abated sufficiently.

CASE 12: HYPERTENSION AND CHRONIC NEPHRITIS.—A woman, aged thirty-eight, who had been pregnant once, consulted us May 11, 1925. Three years before, labor was induced at seven months on account of rising blood pressure (the systolic was 210 at the time of delivery) without edema, headache, or blurred vision. There was pyuria at the time and albuminuria since. The systolic blood pressure varied between 145 and 164.

At examination, the systolic blood pressure was 190, the diastolic 112, the urine contained albumin, graded 2, and pus, graded 1; the hemoglobin was 64 per cent, the phenolsulphonephthalein return was 40 per cent, and blood urea was 26 mg. Examination of the ocular fundi showed mild hypertensive sclerosis. The patient spent ten days in hospital on a diet. The systolic blood pressure was reduced to 138, the diastolic to 98; there was a trace of albumin in the urine, the hemoglobin was 72 per cent, the phenolsulphonephthalein return was 60 per cent, and the blood urea was 24 mg. By water test, the specific gravity of the urine was 1.002 and the output 1175 c.c.; by concentration test the specific gravity was 1.021.

Hypertension with mild chronic nephritis was diagnosed. Further pregnancy was advised against. January 5, 1926, the patient was in excellent health and had no unusual symptoms of any kind.

CASE 13: CHRONIC GLOMERULONEPHRITIS.—A woman, aged eighteen, once pregnant, came to the clinic April 15, 1923. She had had scarlet fever in 1919. There had been a miscarriage at seven months following a convulsion in July, 1922.

When she consulted us, there was a possibility that she was pregnant, although examination of the vagina showed apparently complete atresia. The systolic blood pressure was 176 and the diastolic 112. The average specific gravity of the urine was 1.009; it contained albumin, graded 3, and occasional red blood cells; there was edema, graded 3; there was no return of phenolsulphonephthalein in the test of renal function; blood urea was 158 mg. and hemoglobin was 33 per cent. There were marked retinal changes characteristic of nephritis.

A diagnosis was made of extensive chronic glomerulonephritis in an advanced stage. The patient died June 20, 1923.

CASE 14: HYPERTENSION, ARTERIOSCLEROSIS, AND CHRONIC NEPHRITIS.—A woman, aged thirty-six, consulted us August 15, 1923. Ten years before she had had premature labor at seven and a half months and a dead baby was delivered. She had had dim vision for ten days. There had been two spontaneous miscarriages, one at six months, and one at four months, following a "stroke" during which she almost died. On examination, three months later, the systolic blood pressure was 204 and the diastolic 120; there were residual right hemiplegia and aphasia. September, 1926, the systolic blood pressure was 216 and the diastolic 130, the phenolsulphonephthalein return 55 per cent, the blood urea 28 mg., the eye grounds showed sclerosis of the retinal arteries, and there was no albumin in the urine which had a specific gravity varying from 1.008 to 1.014. At this time she was three months pregnant.

Hypertension, probably of the malignant type, with arteriosclerosis and mild chronic nephritis was diagnosed; therapeutic abortion was advised.

SUMMARY

The classification of nephritis of pregnancy as acute and chronic and the study of renal function in the individual case is an aid to prognosis.

Patients with acute nephritis rarely give a history of previous renal trouble. The presence of albuminuria, the rather sudden rise in blood pressure, and increased edema usually occur during the eighth

or ninth month. Tests of renal function show adequate excretion, aside from oliguria, the specific gravity of the urine is high, and the ocular fundi may reveal evidence of acute changes in the vessels and retina.

Patients with chronic nephritis often give a history of previous nephritis. Albuminuria, hypertension, and edema are apparent earlier in pregnancy, usually before the seventh month. Tests of renal function often disclose retention of nitrogen and phenolsulphone-phthalein. The specific gravity of the urine is fixed or low, and there is definite secondary anemia. Examination of the ocular fundi often reveals changes resulting from previous nephritis.

Most pregnant women with acute nephritis recover with little or no demonstrable impairment of renal function. In some cases the disease will progress to the chronic stage. Many pregnant women with a history of nephritis but with no gross impairment of renal function can be carried safely through pregnancy under careful supervision. Chronic nephritis with seriously lowered renal function distinctly increases the hazard to mother and child.

THE SIGNIFICANCE OF RETRODISPLACEMENTS OF THE UTERUS AND THE PRINCIPLES INVOLVED IN A SATISFACTORY CORRECTION

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PROBABLY I am venturing, when one with more wisdom would hesitate, in bringing before you for discussion a subject that, since the days of the birth of gynecology, has developed more difference of opinion than any other in the field of female pathology. In view of the attention given to retrodisplacements of the uterus and the voluminous literature produced, it would seem that by this time we should have reached a clear-cut vision of the etiology and treatment of this condition, and yet I venture to say that in the discussion to follow, we shall have almost as many views expressed, at least as to treatment, as there are speakers.

For the sake of confining discussion to the subject of the paper, I wish to state definitely that I am not considering procidentia. While a posterior rotation of the uterus, either partial or complete, always precedes a uterine prolapse due to the direction of the pelvic planes, yet the causative factors underlying the two conditions are not wholly the same so that a prolapse may develop in a pelvis where the uterus has previously been in normal position until the time the procidentia appeared. I feel justified in considering this subject in view of the essays that have appeared somewhat recently, wherein the authors ridiculed the condition as being of any moment pathologically and cast

discredit upon those who attempt surgical treatment. One writer speaks of replacing the displaced organ "by gently folding its arms behind its back." Another, as an argument against congestion being produced by the posterior rotation, asks "whoever saw a retrodis-placed uterus ever change its color upon replacement." That such articles have developed the opinion among some internists and others that retrodisplacement of the uterus is of no significance patholog-ically and that correction is unnecessary and even unjustifiable, I am fully persuaded.

That any one pathologic condition can bring forth over 120 types of operation for its correction, I believe shows that we have not yet fully mastered the principles involved or that we are laying too much stress on little modifications that cut no great figure and are thereby causing the average surgeon to lose sight of those principles. Cer-tainly the medical student with such a mass of operative detail to consider must become bewildered and often fail to grasp the funda-mentals.

The men who devised and those who have conscientiously employed one or more of these 120 types of operative correction certainly can-not be credited with doing such work for the sake of fees. Again when benefit to the patient results, we certainly cannot in all cases credit the psychic effect alone with the improvement. Retrodisplace-ments of the uterus would be a thing unknown if women walked on hands and feet, as would any pathology of importance resulting from childbearing injuries.

Nature certainly intended the uterus to lie horizontally in the pelvis, to be free to move within a certain arc so as to compensate for the distended bladder and rectum, and to be able to grow unhampered in pregnancy. Any condition of retrodisplacement or any fixation of the uterus is an abnormal state. That that state should produce abnor-malities of function seems to me to be undeniable. It is perfectly reasonable to expect to find a certain group of women in whom the uterus is small because of underdevelopment, and whose sensibilities perhaps are less acute than the average, who go through life with no consciousness of the abnormality; such are the cases hardly requiring surgical treatment. On the other hand, every honest gynecologist sees many corrected cases cured of their pelvic distress, with the nervous system regenerated in which no other pathology existed. For-tunately we have available a simple method of determining whether such an uncomplicated retroversion is producing symptoms, but un-fortunately this method seems to be falling rapidly into disuse. My experience has been that the younger medical man is not taught or, if taught, does not understand the principles and application of the ret-roversion pessary. If they attempt the use of the pessary, they often overlook the fact that a replacement is the first essential, or they use

a stock form unmindful that many cases require alterations in the curves to fit the individual. I am sorry to say that the majority of the women who have been fitted with pessaries, who drift into my hands, show an improper adjustment with naturally an unsatisfactory outcome.

That the position of the uterus is dependent upon any one factor alone is untenable. There are various factors maintaining the normal position, and each has its own part to play. The structures at the cervical pole support and immobilize that pole. The lateral ligaments prevent rotation and side drift and the fundus supports maintain flexion and allow for elevation but restrain overexcursion. The proper functioning of these various ligaments is dependent to a very vital degree upon the existence of a closed pelvic cavity as maintained by an intact pelvic outlet.

The intact perineum keeps the abdominal and pelvic cavities closed. Thus, under balanced forces and in the resulting closed chamber, the uterus practically floats with even pressure on all sides, except what may be exerted by the variation in cavity contents and respiration. Given, however, an injured outlet, permitting the entrance of air within the vagina, the uterine balance is disturbed, and the cervical structures are forced to bear the strain of support. That a marked relaxation of the perineum with its gradually increasing pull of growing rectocele and cystocele will in time stretch these cervical supports and allow a lowered cervix and an elevated uterus is rationally conceivable, though most retrodisplacements following labor, are hastened by injuries to the structures at the cervical level. A lowered cervix and an elevated fundus must result in a retrodisplacement because of the direction of the pelvic axis and the force lines of the pressure from above. Conversely, it is poor surgery to do any operation for the uterine displacement correction and neglect a relaxed outlet.

With a retroversion, whether secondary to a labor with a resulting relaxed outlet or one that is not so associated, the circulation is interfered with through twisting of the uterine and ovarian arteries and veins, so that the body becomes congested and soft and with the softening a flexion develops.

Once retroverted, the uterus has no tendency to return to the normal for all the pressure from above is on the caudal surface, and the pressure of the bowel contents makes a valve of the fundus. The ligaments are all suspensory and obliged to carry weight for which they were not intended. The tubes are rotated and the ovaries prolapsed, and the condition is aggravated more and more with each developing degree of retrodisplacement. The increased congestion in a body incapable of free expansion, as is the ovary, is bound to produce a tendency to pathologic changes with resultant symptoms.

It is, I think, not unreasonable to believe that the rotations in the fallopian tube may account for some cases of sterility associated with retrodisplacements. Here I wish to sound a warning against tubal inflation in any case of uterine retrodisplacement, without first replacing the uterus. You know what a worn soft garden hose does when the water pressure is turned on and the hose gets kinked. It would not take much pressure to injure the mucous membrane of such a flexed fallopian tube to a sufficient degree to produce inflammation enough to injure that tube permanently. I sound this warning because I have seen some of our obstetricians doing just this thing.

With perhaps the exception of those cases of infantile uterus that are retrodisplaced, a simple case of retroversion does not long remain an uncomplicated one. Even though the ovaries and tubes may not become early prolapsed, there is an interference with their venous circulation due to stretching of the infundibulopelvic ligaments and the twisting of the broad ligaments, so that a hypostatic congestion is produced which tends to develop cystic ovaries. The gradual increase of ovarian weight encourages prolapse. The cervical portion of the uterus is also congested because of twisting of the uterine vessels, and thus there is a tendency to endocervicitis with its end-results of cystic degeneration.

Whether the symptoms so often relieved by a retrodisplacement operation are due to the more normal circulation established in the uterus alone or in the ovaries and tubes alone or in both, is problematic. That only partial relief of symptoms credited to corrected retrodisplacements is obtained can readily be explained by the doing of many types of operation in which the tubal and ovarian elevation is not accomplished. We all know that a prolapsed ovary as an entity has its train of pathology and symptoms and yet many of the advocated displacement operations still leave the appendages low in the pelvis.

In considering the correction of retrodisplacements, I believe it would be well to divide our cases into two classes, separating thus the women who have borne no children from those who have. Such a division would simplify our viewpoint and favor more rational discussion.

The woman with the relaxed outlet and the strained cervical supports, whether the retrodisplacement was primary or secondary to her labors, presents a much more complicated problem. There is a greater lateral elongation of the broad ligaments with more flaccid sacrouterine and vesicouterine structures, although we usually find more body to the round ligaments. The attenuated round ligament is more frequent in the nullipara. In the nullipara unless complicated by some gross pathology, such as tubal or other inflammatory conditions which

have thickened up or stretched out the broad ligaments, the operative correction presents few difficulties, and the maintenance of the forward position requires little tension exerted upon the supporting structures, for there is no downward drag tending to disturb the uterus, and the abdominal pressure helps maintain the posture. Any simple reefing of the round ligaments is sufficient, provided the appendage pull is corrected. Such shortening can be as readily done outside the abdominal cavity if there is no ovarian prolapse and no other pathology exists which requires abdominal entrance, which I acknowledge is not often if the abnormal position is of long duration.

In the woman with a relaxed outlet the primary requirement is, I feel, the proper closure of the pelvic outlet, with the uterine correction secondary. I regret the fact that the tendency of the average operator is to do the abdominal operation and neglect the plastic. If for any reason only one step must be taken and no acute abdominal condition exists, it should be the floor and diaphragm repair. No matter what type of operative uterine correction is done, it will be endangered sooner or later by a relaxed outlet.

In all surgical procedures for the correction of a retrodisplaced uterus, we must recognize first the necessity for an intact, or the production of an intact, pelvic diaphragm and floor. We must not interfere with the free excursion of the uterus within its normal range of motion; this demands that the uterus must not become adherent to the bladder or to the abdominal wall. We must not permit a heavy ovary or ovaries to act as a backward drag upon the fundus; this may even produce a recurrence of the uterine displacement. We must not depend alone upon the round ligaments, no matter how strong, to sustain the uterine position; the broad ligaments have broadened during the period of abnormal position, and the uterine and ovarian vessels have become tortuous.

With these requirements to fulfil, it is evident that the type of operation that fixes the uterus to the peritoneum or abdominal wall is defective through limiting the uterine excursion and producing an abnormal direction for the uterine axis. The type of operation using the round ligament drawn through the abdominal wall is unsatisfactory, because that ligament was never intended for a suspensory ligament and will elongate, also the direction of support is abnormal and the ovarian and tubal drag is still present. In neither type is the broad ligament slack corrected. The plication of the round ligaments and thus, the broad ligaments in front of the uterine body, is a little more rational but has the great disadvantage of a possible bladder fusion, nor does it correct the ovarian prolapse, and it may even distort the arterial and venous uterine and ovarian supply and aggravate uterine stasis.

The type of operation that seeks to reef the round ligaments along their normal course and within the broad ligaments and peritoneal folds presents perhaps less objections as to stasis and abnormal attachments and certainly produces the necessary flexion of the uterus and narrows the broad ligaments, but it does not correct the ovarian prolapse and must be accompanied by an uterine ovarian ligament shortening.

Any operation that confines itself to the plication of the sacro-uterine ligaments alone seems to me hardly worthy of considerations; theoretically, it is a most rational way to elevate the cervix and produce a forward position of the body, but practically the structure of the ligaments does not favor proper fusion and the overloaded lower bowel tends to stretch the shortened tissues.

Finally, the type referred to in my quoting of the satirical reference "to gently folding its arms behind its back" can be an operation which fulfills all the conditions or it may be only a partial success. If the peritoneum is stripped back as the round ligament is drawn through, the broad ligaments are not shortened. If the loop of round ligament is placed too high on the body, too near the fundus, the body can slip under. If the loops are placed too low, the fundus can and will flex over the attachment. Properly done, the operation will correct all the pathology but does produce an abnormal relationship laterally, because of the distorted position of the distal portion of the round ligaments, which depresses the outer portion of the broad ligament, and probably for this reason Webster advised the use of a pessary for some months.

Since 1907 I have been using, when indicated, the Webster operation with my own modifications. First, I do not strip the round ligament from the broad ligament but simply pull the round ligament through the puncture in the broad ligament beneath the ovarian ligament by means of an untied suture loop placed at the desired point on the round ligament. A broad approximation is then made of the round ligament loop upon the posterior uterine surface so as to avoid a rope-like sling; two linen sutures are sufficient. Another linen suture picks up the distal portion of the round ligament about an inch from where it enters the broad ligament and attaches it at the point of insertion of the ligament in the uterus. By this approximation the V-shaped gap in the outer portions of the broad ligament is eliminated. The normal direction of pull is maintained, and flexion is assured. All tension upon the posterior approximation is relieved, and no pessary and no elaborate uterine attachment, as practiced by some, are necessary.

Finally, the test of any operation for retrodisplacement is the cure of symptoms, and the ability to go through labor without recurrence. Many of my patients have had one or more pregnancies, and I have

not as yet met with a recurrence, but this I cannot say for any other type of operation which I have done.

In conclusion, I would emphasize the fact that every patient presents an individual problem and no matter what type of operation is used, it must be modified to meet the conditions present, rather than an attempt made to fit the patient's pathology to the operation, and to accomplish this, the best preparation is a thorough understanding of the fundamentals.

OAKLAND BANK OF SAVINGS BUILDING.

CLOSTRIDIUM WELCHII SEPTICEMIA COMPLICATING PROLONGED LABOR DUE TO OBSTRUCTING MYOMA OF UTERUS, WITH REPORT OF CASE

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CASES of a puerperal septicemia due to the Clostridium welchii are sufficiently rare to warrant a report, especially if positive blood cultures were obtained during life. Forty-five cases have been reported of which 16 gave a positive blood culture antemortem. In the remaining cases, the diagnosis was made upon other clinical, pathologic, or bacteriologic findings.

The purpose of this paper is (a) to report another case of puerperal septicemia due to the Clostridium welchii; (b) to analyze the previously reported cases and attempt to draw therefrom a specific clinical picture, and (c) to review the more recent biologic findings of the Clostridium welchii, especially with reference to their bearing upon puerperal septicemia.

HISTORICAL

The first case of puerperal infection due to the Clostridium welchii is that reported by Leduc[1] in 1597. The woman had been in labor for three days, and after a difficult extraction by means of a blunt hook, a violet colored gas escaped from the vagina. The woman died several days later.

Doleris[2] in 1891 reported a case very similar to our own. The woman was at term, and labor was very difficult, because of a complicating myoma. Forceps and cephalotripsy were attempted, but the fetus could not be delivered. The patient succumbed to a septicemia having its origin in an intense putrefaction of the uterine tissues, physometra, and putrid emphysema of the cellular tissues of the hypogastric region.

Graham, Stewart, and Baldwin[3] in 1893 reported the case of a woman who was seized suddenly with a chill which lasted four hours, and was followed by pain

in the region of the ovaries and uterus, by vomiting and purging. Thirteen hours after the onset she was emphysematous from the top of her head to the soles of her feet, and she died one hour later. Postmortem examination showed gas bubbles in the subcutaneous tissues and a small amount of gas in the peritoneal cavity. The abdominal veins were markedly distended with gas, and numerous bubbles of gas were found in all of the internal organs. The uterus gave evidence of a recent abortion. Bacteriologic examination showed the presence of Clostridium welchii in large numbers.

Dobbin,[5] in 1897, reported the first case in which the diagnosis of puerperal gas sepsis was made during life. The patient had a generally contracted pelvis and had been in labor for two days. On approaching the bed, he noticed a very sweetish offensive odor and heard a distinct continuous crackling sound coming from the vagina. After a difficult instrumental delivery, there was an escape of gas from the uterine cavity accompanied by an explosive sound. The placenta was delivered manually. The patient died four days after the onset of labor, and immediately thereafter the body began to swell. Seven hours after death the body was enormously inflated. From the mouth and nose there exuded a frothy, bloody serum, and from time to time large bubbles of gas could be seen to escape.

Krönig and Menge,[4] in 1894, reported two cases. The first was that of a primipara who could not be delivered with forceps, and cephalotripsy was necessary. There was a foul discharge from the vagina. The temperature remained normal although Clostridium welchii was cultured from the uterus on the third day. On the ninth day the discharge was no longer foul, and the cultures were sterile. The perineal wound failed to heal normally. The fetus showed a generalized gas bacillus infection.

Their second case was that of a woman in difficult labor with prolapse of the cord. She was delivered on account of fever. The lochia was examined on the sixth day postpartum and showed numerous cocci and thick rods. The patient left the hospital on the ninth day. The fetus showed a generalized emphysema.

Little,[6] in 1905, reported 12 cases of puerperal infection, either due to the gas bacillus alone or associated with other organisms. The only case of this series which has any direct bearing on generalized gas infection is his ninth case, the history of which is as follows: febricula, normal labor, isolation of the Clostridium welchii from the uterus, blood culture positive for the Clostridium welchii on the fifth day postpartum, supravaginal hysterectomy, and isolation of the typhoid bacillus and the Clostridium welchii from the blood stream. Death occurred on the twenty-third day postpartum. Autopsy showed typhoid fever, infected abdominal wound, multiple abscesses of the kidney, acute splenic tumor, and healing ulcers of the small intestine. Of the remaining 11 cases of the series, one was Dobbin's case which is reported above; a second was a case of septicemia due to an unidentified anerobic bacillus, and a third was a mastitis caused by the use of improperly sterilized water in hypodermoclysis and from which the Clostridium welchii was isolated. The other eight cases were localized infections of the uterus. Eight cases of the series followed operative procedures, two self-induced abortions, and in one case the organisms apparently entered the blood stream from their normal habitat in the intestines by way of the typhoid ulcers in the intestines. The Clostridium welchii was isolated in pure culture from the breast and from the uterus in two cases, and in the remaining cases it was associated with other organisms as follows: Streptococcus pyogenes, 4; B. coli, 2; Staphylococcus albus, 2; Staphylococcus aureus, 1; gonococcus, 1, and gram-positive diplococci, 1.

Whiteacre,[7] in 1906, reported a case of puerperal gas bacillus infection which followed criminal abortion and which simulated acute appendicitis. The onset was acute with vomiting, fever, diarrhea, and pain in the umbilical region which became localized to the right iliac fossa. At operation, on opening the peritoneal cavity, a considerable quantity of blood-tinged fluid escaped. The appendix was normal.

The patient died three hours after the operation or thirty-nine hours after the onset of the symptoms. The body began to swell one-half hour after death and showed typical gas bacillus changes, postmortem.

Young and Rhea,[8] in 1909, reported two cases which were characterized by a deep copper color of the skin which later darkened to a bronze. Case 1 gave a history of criminal abortion. The onset was sudden with pain in the abdomen, diarrhea, vomiting, chills, and bleeding from the vagina. She was admitted to the hospital on the third day of the disease with symptoms of prostration, restlessness, and vomiting. Physical examination showed a purplish discoloration of the tip of the nose, of the finger tips, and to a less extent of the toe nails. The rest of the body was of a deep bronze color. There was puffiness of the face and extremities; the white blood count was 25,000. Death occurred twenty-four hours after admission. Autopsy showed the usual gas bacillus emphysema. There was present, in addition, a perforation at the fundus of the uterus. The myometrium was dark red, moist, boggy, and crackled on pressure. The uterine cavity contained a considerable amount of grayish colored, seminecrotic material which was thickest and most firmly adherent over an area situated in the upper left portion of the posterior wall of the uterine cavity. The anatomic findings were perforation of the uterus (traumatic), endometritis, metritis, acute generalized peritonitis, and general septicemia due to the Clostridium welchii. Cultures taken from the uterus showed Clostridium welchii, Staphylococcus albus, and B. coli communis. Blood culture which was taken eighteen hours before death was positive for the Clostridium welchii.

In Case 2 the woman started bleeding after heavy exertion six days before admission to the hospital. On the next day the fetus was expelled. On the fifth day after the onset the entire body became jaundiced, and on admission she showed a jaundice with spots of purplish discoloration. The spleen was palpable. Temperature was 98.8° F., pulse 140, and white blood count 126,400. Death occurred four days after admission. Autopsy showed the changes typical of gas bacillus infection. "The uterus was enlarged and spongy to the touch." The myometrium was a light brown, and on section many gas bubbles escaped from it. The placental remains and blood clot were adherent to the uterine wall. The lower third of the endometrium had a distinct greenish hue. Cultures were negative.

Schottmüller,[9] in 1910, reported two cases. In Case 1 there was history of criminal abortion which was followed by chills and bleeding. The following day the patient had three chills with a temperature of 103.6° F. The Clostridium welchii was cultured from the blood and the cervix. The placenta did not have a foul odor. A second blood culture showed numerous colonies of the same organism. There were no further chills, the temperature returned to normal, and the patient was discharged as cured.

In Case 2 there was also history of criminal abortion, followed by bleeding. On the following day the patient had two chills, with a temperature of 100.6° F. Hippocratic facies were present. The Staphylococcus aureus, the pneumococcus, and the Clostridium welchii were recovered from the blood stream and from the cervix. Death occurred at the end of the second day. The placenta was very soft and had a foul odor.

Fraenkel,[10] in 1913, reported a case with the following history: The woman had missed her last menses, and the day before admission she became acutely ill and complained of pain over the abdomen. She showed on admission a peculiar pale gray color of the entire body and signs of peritonitis. Laparotomy, which was performed immediately, showed a large quantity of cloudy, bloody exudate in the peritoneal cavity. At the fundus of the uterus there were two bluish brown tumors, the size of oranges. Hysterectomy was done. The Clostridium welchii was cultured from the uterus and from the blood. Death occurred thirty hours after admission. On the anterior and posterior surfaces of the left half of the body of the uterus

there were present small round tumors which crepitated. On section the ovum and inner lining of the uterus appeared of a dirty brown color, and this discoloration extended almost to the outermost zone of the uterus. The cervix did not show any gas. Methemoglobin was found in the urine and in the blood.

Bingold,[11] in 1914, reported 130 cases of puerperal infection associated with the Clostridium welchii and divided them into the following clinical groups: (a) The localized endometritic type. Here the infection remains localized to the endometrium, is well walled off, usually runs a symptomless course, and practically always ends in recovery, although the organisms may occasionally enter the blood stream by mechanical methods. (b) The lymphangitic type. Here the infection is in the uterine musculature, spreading by way of the lymphatics to the peritoneum and blood stream. This group is always attended with serious symptoms and may end fatally. (c) Thrombophlebitic type. Here the infection spreads by way of the veins and is accompanied by frequent chills. The occurrence of anaerobic streptococci in the latter type would seem to throw doubt upon the Clostridium welchii as being the causative agent in this type of infection.

Cherry,[12] in 1919, reported a case of gas bacillus infection with the following clinical history: Fetal movements were not felt for four weeks. After an interval of four more weeks a Ribes' bag was introduced to induce labor. Thirty-four hours afterward, as no labor pains resulted, a larger bag was introduced. Forty hours after the insertion of the first bag, the patient had a chill and fever. Forty-eight hours later she had an attack of severe nausea and vomiting and suddenly died. Autopsy showed the presence of a dead fetus, and smears from the uterus showed Clostridium welchii.

Matthews,[13] in 1922, reported a case of criminal abortion which was followed twenty-four hours later by bleeding, chills, and vomiting. Forty-eight hours later, on admission to the hospital, the patient was found to be acutely ill, jaundiced, and cyanotic. There was a moderately tender infiltration in the left fornix but more extensive and more sensitive in the right fornix. Red blood cell count, 3,000,000; white cell count, 51,200; polymorphoneutrophiles, 89 per cent; temperature, 104.1° F.; pulse, 120. The outstanding symptoms during her stay in the hospital were numerous chills, cyanosis, and elevation of pulse and temperature. There were marked mental involvement and emphysema of both inguinal regions. Death occurred one hundred and nine hours after introduction of the slippery elm stick. Autopsy showed in addition to the typical changes of Clostridium welchii septicemia, a soft uterus which contained placental remains. Gas bubbles were found over the surfaces of the uterus and broad ligament. Blood culture was positive for Clostridium welchii. Cultures from the uterus gave the Clostridium welchii, B. proteus, and staphylococcus. The peritoneal fluid showed Clostridium welchii in pure culture.

Gemill,[14] in 1924, reported a case of criminal abortion which was followed in three days by severe pain in the lower abdomen and by the passage of a large number of clots from the vagina. On physical examination she was practically pulseless, the brow was covered with a cold sweat, the right arm had red lines over the triceps muscle, and crepitation could be elicited from the elbow to the shoulder girdle and to a less extent on the flexor surface of the forearm. Foul necrotic material was obtained from the cervix. Red blood cell count, 4,865,000; white blood cell count, 10,400; polymorphoneutrophiles, 63 per cent; albumin, 4-plus, and many granular casts. Blood culture was negative. Death occurred five hours after admission. Autopsy showed general emphysema; the spleen was enlarged and soft; the uterus was soft and gangrenous, and contained remnants of placenta.

Lehmann,[15] in 1924, was able to add to a case report details of treatment which saved the patient's life, and shortly afterwards in collaboration with Fraenkel he published it in connection with 4 additional cases.

The following are also included in our statistical study: Welch and Flexner,[16] 1 case; Ernst,[17] 1 case; Halban,[18] 1 case; Lenhartz,[19] 1 case; Stokes and Wright,[20]

1 case; Scheidler,[21] 2 cases; Owen and Glynn,[22] 1 case; Wanekros,[23] 1 case; Rosensohn,[24] 1 case; Hunt,[25] 2 cases; Estol and Hormasche,[26] 1 case; Simon,[27] 3 cases; Heim,[28] 2 cases; Schulz,[29] 1 case; Heim,[30] 1 case; Noltmann,[31] 2 cases; Henyemann,[32] 1 case; Bondy,[33] 1 case; Simonds,[34] 3 cases; Brutt,[35] 1 case; Ford and Lawrence,[36] 2 cases; Coenon,[37] 1 case, and Lehmann and Fraenkel,[38] 1 case.

REPORT OF CASE

C. H. B., colored, aged thirty-two, para i, was admitted to Maternity Division, Memphis General Hospital by ambulance on July 27, 1926, with a history of having been in labor for three days. She was first attended by a midwife for two days who, when labor failed to terminate spontaneously, called a physician. Attempt at delivery by version and extraction was unsuccessful; during the operation, however, the left arm and shoulder were disarticulated and removed.

Upon admission to the hospital examination revealed a fairly well-developed female. Temperature 101.4; pulse 170; respiration 60. She was perspiring freely; there was dryness of the mucous membranes, and cyanosis; she was restless and presented an anxious appearance. No heart tones were audible, and the uterus was in tetanic contraction. Vaginal examination showed a transverse presentation with fetal back over the inlet, the head and right shoulder in the left flank, and the buttocks in the upper right quadrant; the presenting part was crowded firmly against the inlet; the cervix was completely dilated, and the bladder and rectum were empty. The mother was in only fair condition, but delivery seemed indicated, and the method chosen was version and extraction.

Under deep ether narcosis an attempt was made to disengage the presenting part. Relaxation of the uterus could not be obtained, and evisceration was resorted to; the version was then completed without difficulty and was followed by extraction of the body. The fetal head could not be engaged in the inlet; it was evacuated and delivered with the cranioclast.

Immediately after the operation the patient received 1000 c.c. of normal saline solution with 500 c.c. of 5 per cent glucose by hypodermoclysis. Morphine, gr. ⅙, was administered along with digitan and caffeine sodiobenzoate alternately every four hours.

The following morning the patient showed slight improvement. The placenta, which had not been expelled at the time of the operation, was removed manually. Upon removing the placenta which was found closely adherent to the anterior wall of the fundus, a large tumor-like mass was encountered on the left wall of the lower segment which greatly obstructed the inlet. At this time blood and intra-uterine cultures were taken.

The third day postpartum was begun with a transfusion of 300 c.c. of whole blood. The patient's condition was serious. The twenty-four hour blood and uterine cultures showed Clostridium welchii. An hysterectomy was done under local anesthesia, and the patient succumbed at 5 P.M.

Laboratory Findings.—Red blood cells, 3,000,000; white blood cells, 14,506; neutrophiles, 84; lymphocytes, 14; large mononuclears, 2; hemoglobin, 60 per cent; Kahn test negative (7/30); urine on the first day postpartum, showed blood, albumin, pus, and amorphous sediment.

Bacteriologic Findings.—Blood culture taken on July 29 showed within eighteen hours, a gram positive, nonmotile bacillus from the anerobic media. One cubic centimeter of a twenty-four hour broth subculture was inoculated into the ear vein of a rabbit which was killed after five minutes and then incubated for six hours. At this time the rabbit showed bilateral exophthalmos and generalized subcutaneous emphysema. The edematous subcutaneous tissue everywhere contained bubbles of gas, and in areas showed a bright red, greenish, or yellowish discoloration. On entering the peritoneal cavity a large amount of gas escaped. The liver

and kidneys, which were similar in appearance, were opaque, brown, very friable, and filled with gas. The left lung was a reddish brown. The right lung was normal in appearance. The heart had a dull, opaque, brown appearance. Hemolysis, gas, and serum were present in all the tissues, such as muscle, fat, etc. Cultures from the heart's blood and from the liver were positive for Clostridium welchii. The capsule was demonstrated by Huntoon's method. Spores were seen in cultures from alkaline egg media. Milk showed the characteristic, stormy fermentation due to the rapid acidification and coagulation with the production of a large amount of gas. Hemolysis was shown on the blood plate, and inulin and glycerin were fermented, the organism belonging, therefore, to group one.[34]

Cultures from the uterus on July 30 gave B. coli communis, Staphylococcus albus, and Clostridium welchii.

Pathologic Findings.—Only a partial autopsy was permitted. On the day of death the external examination of the body showed a marked generalized subcutaneous emphysema. The conjunctivae were intensely yellowish green. Gas bubbles escaped from the nose and mouth. The vulva crepitated, was swollen, and succulent. The skin was excoriated or came away leaving a hemolytic lower surface. The inner wall of the vagina was gangrenous and of many colors, being similar in appearance to the wall of the uterus previously described.

The uterus was greatly enlarged, weighing 530 grams; the left tube was bound down by a few fibrous adhesions to the surface of the uterus; the right tube was more extensively bound by fibrous tissue to its ovary and to the side of the uterus. There were fibrous tissue tags over the entire surface of the uterus. The uterus was somewhat distorted by the presence of a number of small subserous and intramural myomas, which on section, gave the usual appearance. On being opened the inner surface and the greatest part of the wall and the fundus of the uterus, even reaching the peritoneal covering, was mottled, reddish brown, dark green, yellowish green, opaque, autolytic, friable, and foul smelling. Gas bubbles escaped on pressure. Near the internal os there was a large intramural myoma, 10 cm. in diameter, which projected into the uterine cavity. The outer surface had an icteric tint.

Microscopic examination of the wall of the uterus at the cervix and in the fundus and also of the obstructing myoma showed extensive necrosis of the endometrium and underlying muscle fibers, arteries, veins, nerves, and fibrous tissue. In places were seen small and large spaces which had been filled with gas and in whose wall were seen in large numbers various types of bacteria, including a large round end bacillus, obviously the Clostridium welchii. Serum albumin and extracellular hemoglobin in large amounts were seen between the necrotic tissue fibers. Congestion was marked. The absence of leucocytes was conspicuous. Only small groups of polymorphonuclears were seen in places.

Anatomic Diagnosis.—Puerperal uterus with obstructing myoma and gas bacillus infection.

<div style="text-align:center">DISCUSSION</div>

A study of 41 cases of puerperal septicemia due to the Clostridium welchii which we have selected for detailed analysis, reveals the following facts:

Nature of Pregnancy.—We find that according to the underlying pathology attending pregnancy we can subdivide these cases into: (1) the abortion group comprising 60.97 per cent of the total number of cases, and (2) the nonabortion group, comprising 39.03 per cent. The nonabortion group can be further subdivided into the following subgroups: (a) contracted pelvis 7.29 per cent; (b) obstruction due

to myoma 2.43 per cent; (c) protracted labor 7.29 per cent; (d) death of the fetus 2.43 per cent; (e) transverse presentation 7.29 per cent; (f) breech presentation 2.43 per cent; (g) normal 7.29 per cent; and (h) unclassified 2.43 per cent.

Method of Delivery.—In the nonabortion group we find that the first stage of labor is prolonged in all but 4.87 per cent of the total number of cases. In this group also the pathology which was present in all but 4.87 per cent of the cases was of such a nature that it favored frequent vaginal examinations and various operative procedures. Vaginal examinations and operative procedures serve as the means of introducing into the uterus the Clostridium welchii, which is not normally present in the uterus according to most investigators. Schottmüller,[9] on the other hand, claims that it may be occasionally found in the vagina of normal healthy women and that autoinfection is possible.

Furthermore, both criminal abortions and operative procedures favor the implantation and the multiplication of the Clostridium welchii in the uterine tissues by the trauma and necrosis which they bring about, and it is well known that dead tissue with its deficient supply of oxygen is necessary before the implantation, and especially the propagation, of the organism can be successful.

The prodromal period was found to be within forty-eight hours in 70 per cent of all cases and within twenty-four hours in practically 50 per cent of all cases.

Duration of Sepsis.—This is the interval from the time of infection to the time of death. Recoveries took place in 13.16 per cent, and the majority of these patients had immediate hysterectomies performed; 10.53 per cent died within twenty-four hours; 23.68 per cent died between twenty-four and forty-eight hours; 15.79 per cent died between forty-eight and seventy-two hours; 13.16 per cent died between seventy-two and ninety-six hours; 5.26 per cent died on the fifth day; 2.63 per cent on the sixth day; and the same percentage died on the seventh, eighth, tenth, eleventh, eighteenth, and nineteenth days respectively. In cases where there were many possibilities of infection, we have taken the earliest date as the one of infection, and thus our figures may tend to be higher than actually is the case. Nevertheless, it will be seen that the puerperal septicemia caused by the Clostridium welchii is very fulminating in character. Over one-third of the patients die within forty-eight hours after infection has taken place, and over one-half within four days; but with the early recognition of the disease and the extirpation of the uterus, as shown by Brutt[35] and by Heim,[28, 30] the mortality rate may be reduced considerably. A combination of the extirpation of the uterus (since a debridement is impossible in the case of the uterus) with administration of the specific

serum would seem to us to be the most logical and successful method for the treatment of puerperal septicemia due to the Clostridium welchii.

Symptoms.—In two cases, or 4.87 per cent, the first and only symptoms were those suggestive of air embolism. In Scheidler's[21] case, four days after delivery the patient had sudden difficulty in breathing and died immediately. In Hunt's[25] case, twenty-four hours after performing abortion and while attempting to repeat the act, the patient had a convulsion and died.

Vomiting occurs in 47.37 per cent of the cases and is usually an early symptom; pain in the abdomen occurs in 55.6 per cent; chills in 34.21 per cent; diarrhea in 21.06 per cent, and convulsions in 5.26 per cent.

Pain in the abdomen, especially in the abortive group, is the earliest and most frequent symptom encountered. Vomiting usually occurs at the onset of the disease and then disappears, but occasionally may be present throughout the course; diarrhea may appear early or late.

The skin was described as a grayish blue in 5.26 per cent; as grayish green in 2.64 per cent, and as a pale gray in 2.62 per cent of cases.

Red lines over the triceps muscle were present in one case, or 2.63 per cent.

Puffiness of the face was noted in 5.26 per cent of the total number of cases.

Jaundice occurred in 50 per cent of the total number of cases. It appeared as early as twenty-four hours after infection in 16.6 per cent of the cases showing this symptom. On the second day in 44.4 per cent; on the third day in 5.5 per cent; on the fifth day in 22.2 per cent; on the sixth day in 5.5 per cent, and on the eighth day in 5.5 per cent. It is thus seen that jaundice, when it is present, usually occurs very early in the disease, for 60 per cent of the jaundiced cases showed it within forty-eight hours. It appeared as a mild jaundice which, however, deepened rapidly, so that the descriptions of mahogany-colored, bronze-colored, and dark as a hottentot occurred frequently.

Cyanosis occurred in 42.12 per cent of the cases and usually followed the jaundice, although in one case mention is made of cyanosis without jaundice.

The jaundice is due to the hemolysis of the red blood cells by the hemotoxin liberated by the Clostridium welchii as was first shown by Ford and Lawrence.[36] Fraenkel[10] still maintains that the hemolysis is due to absorption of toxic products from the necrotic tissues. Heim[30] cites a case, in favor of Fraenkel's theory, in which after the infected uterus had been removed, the hemolysis of the red blood cells still continued but ceased after blood transfusion. Cyanosis is due to the lack of sufficient red blood cells to carry on the usual oxygen exchange

necessary for the metabolic processes of the body. Air hunger which is usually present is also due to the same cause.

Jaundice, therefore, is one of the most important diagnostic signs of Clostridium welchii septicemia. A jaundice which appears early and progresses rapidly, and whose final color scheme is a combination of the yellow of jaundice and the blue of cyanosis occurs only in the puerperal infection caused by the Clostridium welchii.

Emphysema of the skin occurred in only 16.59 per cent of the cases. In only 4.74 per cent of the cases was there a generalized emphysema present, such as is seen in gas phlegmon of the extremities. In the remaining cases the emphysema was usually localized and usually terminal.

Changes in the uterus were observed in 13.15 per cent of the cases. In 10.52 per cent there was present tympani uteri and in 2.63 per cent physometra uteri. Thus, it is evident that gas formation in the uterus and in the skin is infrequent, especially when contrasted with the constant occurrence of gas in Clostridium welchii infection of the extremities.

Laboratory Findings.—The urinary findings that are given show that albumin is present in 19.04 per cent, sugar in 4.7 per cent, hematoporphyrin in 4.76 per cent, methemoglobin in 28.56 per cent, oxyhemoglobin in 28.56 per cent, hematin in 9.52 per cent, and hyaline or blood casts in 38.08 per cent. The urine is usually scanty and obtained by catheterization. White blood cells and red blood cells are found in small numbers in the sediment, but in many cases the hemolysis may be so complete that only the shreds of the red blood cells are found. Here again we have the hemotoxin of the Clostridium welchii showing its effects by the laking of the red blood cells, and the excess of the blood pigments excreted by the kidney. Fraenkel[10] noticed a true nephritis in one case, but usually the findings are those of irritation of the kidney parenchyma by the excess of the blood pigments.

White blood cell count: The white blood cell count is between 10,000 and 20,000 in 33.33 per cent of the cases; between 20,000 and 30,000 in 16.66 per cent of the cases; between 30,000 and 40,000 in 8.33 per cent; between 50,000 and 60,000 in 16.66 per cent; between 60,000 and 70,000 in 8.33 per cent; between 70,000 and 80,000 in 8.33 per cent, and 125,000 in 8.33 per cent.

The red blood cell count is normal in 25 per cent of the cases; between 3,000,000 and 4,000,000 in 25 per cent; between 1,500,000 and 2,000,000 in 50 per cent.

In 50 per cent the hemoglobin was between 40 and 50 per cent; in 28.57 per cent between 50 and 60 per cent, and in 42.85 per cent between 20 and 30 per cent.

It is thus seen that the laboratory findings give us very valuable aid in the diagnosis of this infection. In a typical case we should

find scanty, blood-tinged urine containing changed blood pigments, an elevated white blood count, and a rapidly progressive severe secondary anemia. Thus Fraenkel and Lehmann[38] actually observed the red blood cell count sink from 4,200,000 to 3,100,000 to 2,100,000, and the hemoglobin from 70 per cent to 50 per cent to 30 per cent in the course of six hours. Heim[39] emphasizes the finding in the puerperal blood of pathologic bone marrow cells which he claims are only observed in cases of pernicious anemia and gas bacillus sepsis.

Temperature.—The temperature was subnormal in 8.33 per cent of the cases. In 11.11 per cent it was normal, and in the remaining it was elevated, but no definite conclusions can be drawn as to a specific temperature curve.

Bacteriologic Findings.—In 16 cases a positive blood culture was obtained during life. It occurred together with the typhoid bacillus once, with anaerobic streptococci once, with staphylococcus and pneumococcus once, and with B. coli once. In the uterus it was found in 19 of the 41 cases reported. In 9 of these cases it occurred in pure culture; with Staphylococcus aureus in 3 cases; with Staphylococcus albus in 1; with streptococcus (unclassified) in 1; with Streptococcus pyogenes in 5; with anerobic streptococcus in 1, and with B. coli in 5.

The organism occurs in human and animal feces, in naturally fertilized soil, garden soil, street dirt, and on anything that may be soiled thereby, such as clothing, tools, etc. When introduced into the puerperal uterus, it may call forth the following conditions: (1) emphysema of the fetus, (2) puerperal endometritis, (3) physometra, (4) emphysema of the uterine wall, and (5) gas sepsis. Fraenkel groups the first three conditions under the head of tympani uteri and the latter two under the head of physometra uteri. If the condition remains localized to the ovum or to the endometrium and is well walled off, the symptoms may be slight, and the patient usually recovers even though the organism is found in the blood stream. It is, however, when the organism gains entrance into the uterine musculature that the condition becomes serious. In the latter case the organism lodges in the lymph spaces between the muscles and in the lymph spaces of the vessels because of their poor content of oxygen. Then they cause necrosis of the tissues in their neighborhood. Around this necrotic area, we have a peripheral edematous zone due to the extravasation of the blood from the necrotic vessels and its subsequent hemolysis by the hemotoxin. Polymorphonuclears are kept away by a leucocidin liberated by the organism. By way of the lymph stream the organisms are discharged sporadically or continuously into the blood stream, thus giving rise to the picture of gas septicemia. Owing to the oxygen content of the blood, we cannot assume that the organisms multiply there until just before death when with failing circulation the oxygen content of the blood is greatly diminished.

The frequent occurrence of jaundice in gas septicemia and its comparative rarity in gas phlegmon can be explained by the richness of the puerperal uterus in blood and lymph vessels which afford an easy path for the absorption of the hematoxins. The question of the part that the C. welchii and other organisms play in each case must necessarily remain an open one, although there is bacteriologic proof that in one case at least the C. welchii died out and death was caused by another organism.

CONCLUSIONS

1. A summary of the recorded cases shows that abortion is the most important cause in the etiology of gas bacillus infection.

2. Pain in the abdomen, fever, rapid pulse, jaundice, cyanosis, air hunger, hemoglobinuria, hemoglobinemia, rapidly progressive secondary anemia with pathologic bone marrow cells in the circulating blood form the clinical entity for the diagnosis of gas bacillus sepsis.

3. Hysterectomy, administration of antitoxin, and blood transfusion seem to be the most logical method of treatment.

We are indebted for assistance in this study to Dr. Harry C. Schmeisser, director of the Pathological Institute, University of Tennessee.

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IS THE EXPECTANT PLAN OF TREATING HYDATIDIFORM MOLE JUSTIFIED?

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CYSTIC degeneration of the villous processes of the chorion, commonly called hydatidiform mole, is the most frequent, as well as the most dangerous morbid alteration of the ovular envelop. This rather unusual type of neoplastic disease is almost invariably accompanied with an embryonal death rate of 100 per cent, and one patient out of every eight or ten dies, either as a direct result of the associated hemorrhage or indirectly from chorionepithelioma, the most fatal type of epithelial malignancy.

Though placing the maternal mortality of hydatidiform mole at a relatively low point, I am not unmindful of the much higher figures set forth in the standard works on gynecology and obstetrics. The recorded maternal mortality is consistently fixed at from 20 to 25 per cent and, in the final analysis, these figures may not be far from the mark.

Notwithstanding a discrepancy in statistics of 5 per cent or more, it is obvious that no other disease complicating pregnancy, except perhaps septic infection, is accompanied with a higher percentage of maternal deaths.

It is highly probable that chorionic malignant disease rarely, if ever, arises independently of hydatidiform mole, although molar degeneration may not be recognized or even suspected.

Probably a larger number of moles than the accepted 5 per cent become malignant. Primary malignant degeneration of the placenta is exceedingly unlikely, and I am persuaded that hydatidiform disease is the precursor of chorionic carcinoma in nearly all cases.

Typical chorionepithelioma follows some type of pregnancy invariably, and in the majority of instances, as is well known, it follows vesicular mole.

The next condition in point of frequency is abortion, but this statement must be accepted with some qualification. It is extremely important to determine whether those cases designated as abortion fulfilled completely the condition embodied in that term. Were all cases so recorded, thoroughly studied from the gross as well as the histologic aspect? Is it not likely that many of the cases were casually recorded as simple abortions? Is it not possible that some contained concealed or microscopic evidence of early vesicular degeneration?

Meyer believes that hydatidiform mole is an extremely uncommon condition at or near term, but he regards it as the most frequent of

all diseases of the ovum during the early weeks of pregnancy. This investigator claims that from 4 to 10 per cent of all pregnancies are complicated by some degree of hydatidiform degeneration. The determination of the frequency of the trouble, he claims, depends on the care with which all specimens are examined.

As regards the malignant transformation of hydatidiform mole, Teacher found this disease arising after mole in 36.6 per cent and after abortion in 31 per cent of cases. In 240 cases of chorion epithelioma, Hitchmann and Cristofoletti found malignancy following mole in 48.3 per cent and after abortion, in 73 per cent of cases. These observations, referred to by nearly all writers, were made more than twenty-five years ago, and they are not without significance.

In view of the extensive investigations and findings of Meyer, it seems rational to assume that many of the cases simply registered as abortions were in reality early moles. It is almost inconceivable that a simple uncomplicated abortion could act as the progenitor of malignant disease.

In this connection, it may be of interest to recall that nearly all cases of mole are treated primarily as threatened or inevitable abortions. The few recorded cases of chorionepithelioma developing during pregnancy may represent the sequel of primary hydatidiform mole, and this may likewise apply to those rare cases following tubal gestation.

In a personal case of the latter type, I was able to demonstrate without difficulty numerous small cystic bodies in the tumor mass, and in studying a similar case of a colleague, I found plain signs of associated cystic disease. Most observers contend that hydatidiform mole, so far as malignant degeneration is concerned, does not reach the figure of 5 per cent, or the maternal mortality the high point of 20 or 25 per cent as recorded in the literature. Why this attitude is assumed, I am unable to understand, for I am convinced that the figures mentioned are in almost strict accord with the clinical history of the disease.

Since this paper is designed primarily to consider the treatment of hydatidiform mole, many other interesting phases of the trouble cannot be included in this discussion. In order, however, to present the subject in a systematic manner, I have found it necessary to incorporate certain features of the condition besides its treatment.

With reference to the historical aspect of hydatidiform mole, it might be of interest to mention that, while the disease was first described by von Grafenberg in the latter half of the sixteenth century, special interest was not aroused until the publication of the paper of Madame Boivin, one hundred years ago, 1827. However, it was not until the beginning of the nineteenth century that the lesion was finally recognized as a degenerative process of the chorionic villi.

There is now, as Schumann states, "a rich and voluminous literature relating to the genesis and pathology of these growths, though the management of the condition has excited but scant comment."

With reference to the etiology of the disease, nothing is definitely known. Many predisposing causes, as age, time of occurrence, multigravity, and the association of lutein cysts are mentioned, but the actual causation of the trouble is wholly obscure.

With our present knowledge regarding the association of lutein cysts, structure at times assuming an attitude of aggression, at other times, one of regression, it is impossible to determine whether their presence be incident to the pregnancy or coincident to the pathologic alteration in the chorion. That lutein cysts may be etiologically related is possible, but thus far no legitimate proof that they are actually causative has been presented. To stand the test of etiologic relationship, they should be at least a fairly constant and not an occasional accompaniment of hydatidiform mole or its most common sequel, chorionepithelioma.

Hydatidiform disease is still looked upon as one of the uncommon complications of pregnancy, but it must be admitted that the trouble is much more frequent than formerly believed. Opportunity is not afforded many individuals to study a large series of cases. Even for one in active obstetric work opportunity for study is limited. Probably not more than ten or fifteen cases of the gross type of the disease come under one's observation, yet it is not too much to say that one or more out of the number thus observed terminate in chorionepithelioma.

A study of the literature of so-called simple benign mole with reference to its frequency discloses a wide gap. Accurate figures respecting its incidence are not forth coming, but modern writers believe the condition occurs with much greater frequency than was formerly taught.

Madame Boivin, writing in 1827, to whom nearly all authors refer, claimed the disease arose once in twenty thousand pregnancies.

Freund placed the complication at a similar figure. Broadhead and Kassebohm, in a study of 12,030 cases of labor and abortion, found six moles, or one in 2,000. Gordon, in a study of 4,500 abortions in Bellevue Hospital, New York, found twenty-one cases of mole, or one in 214. In a series of 348 pathologic abortions, this writer found hydatidiform degeneration in 43 per cent.

In 8,187 maternity patients, Velasco discovered forty typical examples of hydatidiform mole in 205, or 5 per cent.

Meyer, in analyzing 2,589 abortions in the Mall collection, found on gross examination alone eight specimens of mole, or one in 261 cases.

Storch, quoted by Frank, in an examination of a large series of unselected abortive ova, found 75 per cent showing cystic degeneration. A study of the recent literature indicates a growing conviction that the condition is more frequent than heretofore believed.

Reference has already been made to the relationship of hydatidiform mole and chorionepithelioma, and to no subject in gynecologic or obstetric pathology, except perhaps uterine cancer and puerperal infection, has more critical investigation been devoted. As a result a voluminous literature has arisen, especially with reference to the influential rôle hydatidiform mole bears to malignant degeneration of the placenta.

Up until 1921, there were recorded in the literature 587 cases of this type of malignant disease, and in a comparatively recent study covering a period of three years, I found recorded twenty-five additional cases, making a total of 612. These figures, I am quite confident, do not represent by any means the total number of cases that have occurred.

Pallosson and Violet, in studying the relation of hydatidiform mole to chorionepithelioma, found 203 cases, or 44 per cent, in 455 specimens examined.

Of 500 cases of mole collected by Findley, 157, or 31.4 per cent, became malignant.

Kerr believes hydatidiform mole precedes chorionepithelioma in 50 per cent of the cases, and of Teacher's well-known series of 188 cases, 74, or 39.3 per cent, were preceded by molar disease.

The mortality of vesicular mole depends: first, somewhat on the stage of pregnancy at which the tumor begins; second, on the invasive nature of the growth and, third, on its benign or malignant character.

In the 500 cases collected by Findley, there were 265 designated as benign; of this number 237 of the patients recovered and 28 died, a mortality of 10.5 per cent. Ninety-nine patients developed secondary malignancy. Forty-five of these recovered and fifty-four died, a mortality of 54.5 per cent.

In a series of sixty-eight cases of mole recently recorded in the literature, the ultimate result was mentioned in sixty-four. Fifty-eight of the patients recovered and six died, a primary mortality of 9.4 per cent. The cause of death was attributed to hemorrhage in four, two of which were associated with perforation and rupture of the uterus. One patient died of peritonitis and one of secondary chorionic malignancy.

With regard to the mortality rate as indicated in the literature, it is figured as low as 9 per cent by Gordon and as high as 26 per cent by Williamson. Provided all patients receive intelligent care, probably a primary mortality of 10 per cent could be looked upon as a fair minimum and 15 per cent as a fair maximum, exclusive of secondary malignancy.

Treatment.—Although active measures in the treatment of hydatidiform mole have from time to time been advocated, a policy of tardy expectancy is still usually adopted. This rather complacent attitude has not altered during the last half century. A disease associated

with a primary mortality of 10 or 15 per cent with an added mortality from secondary malignancy calls for more intelligent, if not more drastic therapy.

Schumann, in a paper published in 1922, claims it is rational to regard every hydatidiform mole clinically as malignant, to which no halfway measures are applicable. He advocated removal of the uterus with the tumor through an abdominal incision. This plan has not met with approval. Schumann does not stand alone as the sole proponent of the radical method, nor was he the first to bring it to the notice of the profession. Many years ago the procedure was suggested by Freund. It has been approved by Essen-Möller and supported by Howard Taylor, especially in those cases associated with bleeding, in patients with rigid cervices and those approaching the menopausal years.

In extenuation of the radical plan advocated by the writers named, it must be said that they are largely justified in their premise, since no other condition complicating pregnancy carrying a like mortality would be treated by sheer complacency, the means commonly employed today.

The expectant method of treatment is not founded on a scientific basis. *Expectancy is almost analogous to helplessness. Tardiness has not served to mitigate the primary mortality of the disease nor thwart the development of secondary chorionepithelioma with its widespread metastasis. No disease of a potentially malignant nature, one of the chief characteristics of hydatidiform mole, is amenable to a policy of watchful waiting.*

While personally I am not prepared to adopt the ultra radical plan suggested by the writers named, I am nevertheless fully convinced that the present mode of therapy needs revision. It should be governed, it seems to me, as other forms of neoplastic disease, by the revelations of the microscopic and not by the subsequent clinical course of the growth.

Every mole expelled should be followed by a careful curettement of the uterine cavity. This recourse should be instituted irrespective of whether the expulsion be partial or complete. The material removed, as well as that expelled spontaneously, should be subjected to the most exhaustive microscopic. examination.

Abnormal activity in Langhan's cells (Fig. 1) should constitute the guide for further treatment. Simply curetting the uterus combined with a course of wait-to-see-what-may-happen does not provide the necessary factor of safety.

To be governed by recurrent or persistent bleeding, a custom practiced hitherto, is not scientific nor is it justified. The advent of secondary hemorrhage is always exceedingly significant, if not really ominous. Recurrent bleeding may be an expression of a hyperplastic

process in the endometrium, but in some instances it is an expression of secondary malignancy. In still other cases it may be due, as pointed out in a very splendid paper by Rosenzweig, first, to syncytial endometritis and, second, to syncytioma.

Although it is said there is no cell type of hydatidiform mole to indicate its benign or malignant character, evidence is gradually accumulating to show that this conception is somewhat in error.

Doctors Crawford and Bucher, pathologists in Jefferson Medical College Hospital, from studies of the specimen expelled by the patient,

Fig. 1.—Section from an expelled mole, showing the presence of villi and marked proliferation of the epithelium.

Mrs. A. F. S. (Fig. 1), made a tentative diagnosis of incipient chorionepithelioma. These microscopists were so positive as to the nature of the growth that they even recommended immediate hysterectomy. This advice was not heeded. The usual plan of wait and watch was followed.

Four weeks after expulsion of the mole the patient again came under observation complaining of discomfort about the vaginal orifice. An examination disclosed two masses the size, color, and consistency of the common blue plum. One of the tumors occupied the

midsection of the anterior vaginal wall, and the other was located immediately within the vulvar orifice in the posterior vaginal wall.

This patient was readmitted to the hospital and a hysterectomy was performed, the vaginal growths being removed at the same time by means of the cautery knife. The vaginal tumors, were submitted to the pathologist and were reported as typical examples of chorionepithelioma. (Fig. 2.) A microscopic examination was made of several sections of the uterine body, but curiously this did not show more than an atypical syncytial invasion of its wall.

Fig. 2.—A portion of the growth removed from vaginal wall.

This single example of rather characteristic cellular alteration in a hydatidiform mole would seem to disprove the assertion of certain writers, notably Caturani, that there is no cell type to distinguish the premalignant growth, if I may use that very much abused term. Doctors Crawford and Bucher regarded the growth as approaching the malignant type, basing their histologic diagnosis on the extraordinary activity of Langhans' cells.

In this connection it may be important to recall that Caturani, who has devoted considerable study to the clinical aspect and cellular architecture of moles, refers to certain features which might enable one to determine their true oncologic nature. Specimens in which

features of the primitive chorion are reproduced in the cystic mass, he always views with suspicion. Signs of a malignant propensity are also indicated to a certain extent, in those tumors displaying a tendency to invade the myometrium. The latter types hitherto regarded, I believe, as the transitional stage of a benign mole to a malignant chorioma are now, according to Ewing, Rosenzweig, and others, looked upon essentially as benign growths.

This observation may explain those rare instances of spontaneous cure of certain growths recorded as malignant and characterized by perforation of the uterine wall, bladder infiltration and, even pulmonary metastasis. .

Finally, is the customary plan of expectant treatment of hydatidiform mole justified? From the varied clinical and pathologic aspects of the disease, this question must be answered in the negative. To meet the premise of sound therapeutic philosophy, the means employed should be governed by the judgment of an expert pathologist. It should be based on the pathology rather than on the clinical behavior of the growth.

The following abstracts of ten case histories collected from our hospital files portray some of the clinical and pathologic features of hydatidiform mole.

CASE 1.—Mrs. M. D., aged thirty, nullipara, admitted to Jefferson Hospital with a history of irregular bleeding following an amenorrhea of three months' duration. Forty-eight hours after admission she expelled a large mole. This was submitted to microscopic study and was reported benign. Two months later the patient returned to the hospital complaining of more or less persistent bleeding and a diagnostic curettage was performed.

The tissue thus removed consisted of organized blood and a few grape-like cysts. From the fragmentary character of the blood combined with the cystic bodies, it was thought wise to perform an exploratory hysterotomy. On exposing the interior of the uterus, there was found in the left posterolateral wall a necrotic mass three centimeters in diameter. The uterine incision was closed and a complete hysterectomy was immediately performed. Section of the uterine body disclosed several small cysts penetrating the myometrium, and two of these were found hanging from a perforation in the left uterine wall. Sections of the tumor were studied, and a diagnosis of chorionepithelioma was returned.

Nine months later, the patient again came under observation complaining of vague pain in the left side of the pelvis. On bimanual examination a soft globular mass, the size of a small orange, was located in the left pelvic cavity. An exploratory posterior vaginal incision was made. Manipulation of the tumor provoked violent hemorrhage, from which the patient almost immediately succumbed. Some of the tissue, grossly resembling organized blood, removed through the posterior vaginal incision was submitted to microscopic study and proved to be, as was suspected, recurrent chorionic malignant disease.

CASE 2.—Mrs. F. S., aged thirty-seven, para i. The patient was admitted with a history of irregular bleeding following an amenorrhea of three months' duration. Bleeding began after an eight weeks' amenorrhea. Twelve hours after admission, the patient expelled a large hydatidiform mole. Microscopic examination of the specimen disclosed extraordinary activity of Langhans' cells. The pathologist looked upon the tumor with grave suspicion, and advised, though did not urge,

a hysterectomy. The operation, however, was postponed. Four weeks subsequently, the patient returned to the hospital complaining of distress in the vaginal canal. Examination revealed two plum-like growths, one in the anterior and the other in the posterior vaginal wall.

These tumors together with the uterus were removed. On microscopic examination the growths proved to be typical chorionepitheliomas. The patient was operated upon in June of the present year and thus far there is no evidence of recurrent disease.

CASE 3.—Mrs. N. G., aged thirty-five, para vi. The patient was admitted to the hospital with a history precisely similar to that of patient number one, though the hydatid mass primarily expelled was regarded as an abortion. In this patient a large globular mass was found in the anterior vaginal wall. In the uterine body there was a spherical mass, measuring twelve by six by four centimeters. The uterine body together with the vaginal tumor was removed. Microscopic examination showed both the vaginal and uterine growths to be typical chorionepitheliomas. This patient made a good recovery and has not developed recurrent malignant disease.

CASE 4.—Mrs. E. L., aged twenty-six. This patient had a vaginal hysterectomy performed for chorionepithelioma. Recovery. No recurrence.

CASE 5.—Mrs. G. E., aged twenty-five. This patient had an abdominal hysterectomy for chorionepithelioma and recovered. No report of recurrent disease.

CASE 6.—Mrs. L. M., aged thirty-five, para iv. Expelled a large mole. She was curetted twice before admission. An abdominal hysterectomy for chorionepithelioma was performed. The patient died eighteen days later of widespread metastases with pulmonary deposits especially conspicuous.

CASE 7.—Mrs. S. B., aged forty-two, had a dilatation and curettement for a large hydatidiform mole. Death followed from hemorrhage.

CASE 8.—Mrs. M. D., aged thirty-five, had a dilatation and curettement for a hydatidiform mole. No report of recurrent disease. Recovery.

CASE 9.—Mrs. E. P., aged twenty-nine, para ii. This patient had a dilatation and curettement for a hydatidiform mole. Died a few hours subsequently of severe bleeding.

CASE 10.—Mrs. M. U., aged seventeen. This patient had a dilatation and curettement for a hydatidiform mole. Recovery. No report of recurrent disease.

This series of ten cases may convey some idea of the gravity of chorionic villous disease. Six of the patients developed chorionepithelioma. Four recovered and two died. Four of the patients had simple moles. Two recovered and two died.

A STUDY OF PREGNANCY AND PARTURITION IN PRIMIPARAE WITH BICORNUATE UTERI

By Frederick Howard Falls, M.S., M.D., Chicago, Ill.

(From the Department of Obstetrics and Gynecology, University of Illinois, College of Medicine)

PREGNANCY occurring in bicornuate uteri of more marked degrees, such as the uterus didelphys, uterus duplex, and the uterus with a rudimentary horn has been observed and frequently recorded. Many isolated cases in the literature deal with serious dystocia occurring in those cases.

There is little or no mention, however, of pregnancy and parturition occurring in the milder type of bicornuate uterus, such as the uterus septus, subseptus, and arcuatus. Indeed little attention is paid to these deformities beyond mentioning their existence by the authors of various textbooks. From our observations during the past six years, we feel that anomalies of this type are not uncommon and that they frequently give rise to unwelcome complications, some of which might be avoided completely or their danger minimized if the diagnosis of these deformities were more often made and their significance appreciated.

This study is based on a series of cases observed during the past six years. I would like to call your attention to certain signs and symptoms that seem to be characteristic as well as to show the differences between individual cases and to point out the possible dangers to the mother and fetus inherent in these cases.

The frequency of these deformities is estimated to be about one per cent. I have noted the condition about equally frequent in the white and colored races. No other deformities were noted in these women. Twin pregnancies or other anomalies of development were not especially found in their family histories.

The essential uterine pathology varies with the degree of deformity. Unfortunately it has been difficult to study the essential pathology of the uterine wall and its placental attachment except grossly at operation. We have had one autopsy following a cesarean section, and the bicornuate nature of the uterus was observed.

The essential points seem to be a thinness of the uterine musculature, especially in the lower uterine segment, with rotation and disproportionate enlargement of one or the other horn. Small infarcts of the placenta and a tendency to its anomalous contour (placenta biparte) are frequently seen.

Associated pathologic findings may be upward displacement of the bladder. The bladder was found reaching almost to the umbilicus in two cases. Oblique presentation of the fetus which may be almost

transverse in some cases and intrapartum fetal death from asphyxia may occur. There does not seem to be any tendency to other anomalies of development or deformities in these women as far as I could observe in this small series.

The signs and symptoms presented by patients whose uteri are of this type are characteristic and not difficult to recognize if looked for.

Inspection frequently reveals the uterus deviated to one side of the midline. This is frequently supplemented by the impression of unusual breadth of the fundus of the uterus. In some cases, especially during a Braxton-Hicks contraction, a notch may be seen between the two horns of the uterus. At operation the notch between the horns can be clearly seen after the delivery of the baby.

Percussion corroborates the above findings and may be of considerable help in outlining the uterus, especially in stout women. Auscultation is one of the most important of the diagnostic signs. The fetal heart tones are frequently found to be irregular in rate and strength and may be either too fast or too slow, or what is more common, alternating fast and slow during the count for a period of a minute. Rates of one hundred and sixty and one hundred and seventy even before the patient goes into labor have been noted. Palpation gives, in a woman with thin or moderately thick abdominal walls, the most important diagnostic aid. Often the fundus will be felt to extend from one anterior axillary line to the other. The position of the notch and its depth varies. If one horn is larger than the other, the notch is usually eccentric and shallow; when both horns are of equal size, the notch is usually more pronounced and more central. I have not observed any of these patients in the early weeks of pregnancy. I have under observation at this time a patient who at about six weeks of pregnancy had an angular pregnancy or *grossesse angulaire*. She is now about four months pregnant, and there is some evidence that the uterus may prove to be of bicornuate type. The deviation of the uterus from the midline and its rotation on its longitudinal axis can also be made out. In some cases an oblique presentation due to the displacement of the presenting part into an iliac fossa and the location of the opposite pole of the fetus in the other subcostal region may be made out. This is often spoken of as a transverse presentation and in a primipara is very significant.

Failure of the head to engage at term, and a tendency to premature or postmature delivery is common. In one patient with a complete uterine septum, the impossibility of forcing the head to engage was a marked feature. Postpartum examination revealed a broad fundus in several cases and in one or two a notch could be palpated.

A résumé of the clinical course of labor in these cases is shown in Table 1.

Postmaturity, two weeks or more, was noted in four out of the fifteen cases. Only one of this series delivered prematurely at the thirty-fifth week. The average age of these primiparae was twenty-six years, the oldest being thirty eight and the youngest fifteen. Six of the fifteen cases that were allowed to go into labor had a labor of more than seventeen and a half hours, the longest being forty-one, and this was terminated by cesarean section. The average blood loss was estimated to be about five hundred cubic centimeters, which is about twice as much as the average blood loss for a normal case in our clinic. I have felt that this was probably best explained on the basis of a faulty contraction and retraction of the uterine musculature in these cases. Involution was found to be slow in one-half of the cases and normal in the rest.

A study of the babies in this series showed them to be normal in size, weight, and development for the period of gestation. Both babies that died in utero were autopsied, and no cause for the death other than asphyxia could be found. Irregularity of the fetal heart tones in eight out of the fifteen cases was noted. In some of these cases irregularity of the rate was the only deviation from the normal, while in others the fetal heart tones were unusually rapid (180) or unusually slow (100).

Labor terminated spontaneously in six of the fifteen cases. Four cesarean sections were done, two versions and extractions, one breech extraction, and two forceps. Labor was started by bag induction in five cases. The placenta had to be removed manually in two cases. Four of these cases were preeclamptic toxemias. Two mothers died following cesarean section, one had been in labor forty-one hours and the other thirty-six hours when they came to operation. They entered the clinic after several hours of labor and after having had some vaginal examinations which were said to have been carefully done. The autopsy showed infection of the uterus and general peritonitis. Two babies died in utero, one before labor started and one during the first stage, before any dilatation of the cervix had occurred.

Treatment in these cases demands considerable judgment on the part of the obstetrician. The greatest concern during pregnancy should be for the child. Heart tones constantly above one hundred fifty and one hundred sixty should be carefully and frequently observed. If the rate continues rapid and the baby is near term, I believe delivery should be accomplished either by induction of labor or by cesarean section, depending on the degree of irregularity and whether or not labor is complicated by other serious obstetric conditions, such as toxemias or pelvic malformations. The tendency of these patients to go over time should be kept in mind, and labor should be induced before the baby has become too large. In this connection due allowance should be made for the primary uterine inertia which is

TABLE I

NO.	AGE	MENSES	TERM	LENGTH OF LABOR	TYPE	DIRECTION OF DEVIATION	HEMOR.	INVOLUTION	WEIGHT OF BABY	LENGTH OF BABY	F.H.T	B.P.	RESULTS	REMARKS
1	19	14 Reg. Mod.	Plus 12	12.5 hr. 3.5 hr. 9 min.	LOA Spon.	¾ Rt.	200	O.K.	3800	52.5	124 178	124 78		
2	15	12 Reg. Mod.	Plus 4	41 hr.	Ces. sect.	¾ Lt.	450	O.K.	3740	48.5	Irr. 140		M. d	Bladder at umb. Def. notch after delivery
3	32	13 Irr. Pain	Plus 15	8 hr. 15 min. 9 min.	LOA Spon.	Rt.	150	O.K.	3315	50	144 160	158 105	B. d	Preeclamptic
4	18	13 Reg. Pain	0	19 hr. 35 min. 12 min.	LOA Spon.		275		2870	50.5	140	180 90		
5	24	11 Reg. Prof.	Minus 1	13¼ hr. 2¾ hr. 11 min.	LOA Spon.		100	O.K.	3200	49.5	100 164	178 86		Bag induction Preeclamptic
6	37	14 Reg.	Plus 2	36 hr.	Ces. sect.	Rt.	500	O.K.	3230	50	Irr.	140 78	M. d	Contracted pelvis Bladder high
7	18	14 Reg. Mod.	Plus 5	16½ hr. 52 min. 4 min.	Ext.	Rt.	1500	O.K.	2380	46	Irr. 124 132			Bag induction Breech delivery Manual removal pl.

TABLE I—CONT'D

NO.	AGE	MENSES	TERM	LENGTH OF LABOR	TYPE	DIRECTION OF DEVIATION	HEMOR.	INVOLUTION	WEIGHT OF BABY	LENGTH OF BABY	F.H.T	B.P.	RESULTS	REMARKS
8	24	15 Reg. Mod.	Minus 38		Ces. sect.		350	Slow	2280	44.5	Reg. 140	112 68		Pyelitis Prem. sep. of plac.
9	21	14 Irr. Scant	Plus 2	13 hr. 45 min. 10 min.	LOA Spon.		300	Slow			Reg.			Bag induction
10	31	13 Reg. Prof.	Plus 17	15 hr. 5 hr. 14 min.	LOA Spon.		200	Slow	2625	51	Reg. 140	100 60		
11	18	14 Reg.	0		Forceps		2000		3100	50	156 180			Intra and postpartum hemorrhage
12	37	14 Reg. Mod.	Minus 4		Vers. ext.		300	Slow	2340		Reg.			Pulmonary Tbe Preeclamptic Bag induction
13	28	13 Reg. Mod.	Plus 3		Ces. Sect.	Rt.	250	O.K.	4044	51	Irr. 130 100	110 60		Contracted pelvis Pyelitis
14	38			34 hr. Weak pain	Vers. ext.	Rt.	800	O.K.	3045		130 160		B. d	Bag induction
15	26	Irr.	Minus 14	30 hr. 1 hr.	Forceps	Rt.	250	O.K.	2840		Reg. 160			Bag induction Preeclamptic

so commonly seen and which is probably due to the faulty muscular development. Preparations to combat postpartum hemorrhage and for manual removal of the placenta should be made. Because of the frequency of fetal asphyxia, the proper facilities for resuscitation should be provided.

In those cases in which delivery from below is elected, the fetal heart tones should be carefully observed throughout labor, especially in the second stage, and arrangements for operative delivery by version and extraction, breech extraction, and forceps should be made. In those cases in which cesarean section is elected, the high position of the bladder should be remembered and the tendency to torsion and lateral deviation of the uterus should be kept in mind. Cesarean section is more frequently advocated in elderly primiparae showing this deformity, than in women of the same age with normal uteri, because of danger to the child in the primary as well as in subsequent labors.

<center>CASE NOTES</center>

The first patient showing this anomaly came under our observation as a private patient in 1920, primipara, thirty-eight years of age, whose menstrual history was without abnormality. She entered Lying-in Hospital one afternoon about 1 P.M., having had weak pains all morning. These pains persisted throughout the afternoon. During the latter months of pregnancy we had noticed that the head was deflected to the left and rested more over the iliac fossa than over the inlet. We also noted that the heart tones were unusually rapid, varying from one hundred and fifty-six to one hundred and sixty-six. This rapidity together with some irregularity following her entry into the hospital was especially noticeable when we tried to force the head over the inlet and into the pelvis. The pains progressed, and the heart tones were present at 6 P.M. Pains were weak, and the patient was making very little progress. The next observation was at 7 P.M.; the heart tones were absent and a definite diagnosis of the death of the baby was made. Labor was allowed to progress the next day and by nine the next morning, since there was practically no advance in spite of weak pains occurring about every ten or fifteen minutes, a Voorhees No. 4 bag was inserted through the cervix. Stronger pains then came on and the bag was expelled about 4 P.M. that afternoon. Following this the pains kept up and the cervix went to nearly complete dilatation; by nine-thirty the patient was showing signs of exhaustion, and there was some evidence of threatened rupture of the uterus. She was, therefore, anesthetized and delivered by version. On introducing the left hand into the uterus, it was found that there was a septum between the examining hand and the baby, though the hand was in the cavity of the uterus. A diagnosis of a bicornuate uterus with septum was made, the septum was perforated, and the baby delivered by version. The patient went into severe shock after the delivery, but rallied and made an uneventful recovery. About a year and a half later I received a letter at the University of Iowa from Dr. DeLee, saying that he had performed a low cervical cesarean section on this patient and had been able to demonstrate at operation the septum which I had penetrated previously.

The second patient of this type was seen at the University of Iowa and presented a ten pound baby with a generally contracted pelvis. The heart tones were noted to be between one hundred and fifty and one hundred and seventy on numerous examinations. There was a deflection of the uterus to the left, so that about seven-eighths of the uterus was to the left of the midline. Because of the size

of the baby and a postmaturity of about two weeks with abnormally, and rapid heart tones, it was felt that the safest method of delivery was cesarean section. At the operation the hypertrophied left horn of the uterus was plainly evident, and the right horn was presented by a slight elevation on the surface of the hypertrophied left horn after the baby was delivered. The round ligament and tube were attached to this small horn.

A third patient was seen at the Cook County Hospital under the following circumstances: The interne on my service reported that he had found a transverse presentation in a primipara with the heart tones one hundred and seventy, and the patient was having strong pains. Having in mind these babies that died in utero, I told him to reserve the operating room for possible cesarean section, and asked him to have an x-ray picture made immediately. I arrived at the hospital just as the patient was placed on the x-ray table for the taking of the picture. Just as the exposure was being made, the membranes ruptured, and the plate was ruined. I listened to the heart tones immediately, and they were not detectable. Without waiting for another picture, we transported her rapidly to the operating room on the floor above. I scrubbed up hurriedly, had the patient rapidly prepared and draped, and did a version and extraction. Following the delivery of the baby I introduced my hand into the uterus and the arcuate nature could be very clearly demonstrated, by inserting the hand into the horns. The baby was resuscitated with some difficulty.

CONCLUSIONS

1. Mild degrees of bicornuate uterus (uterus arcuatus) are not very uncommon.

2. They tend to produce oblique presentations which in primiparae are presumptive evidence of the deformity.

3. The uterus tends to deviate to one side of the abdomen in many cases.

4. Unusual breadth of the fundus or a notch in its upper surface is frequently seen.

5. Prolonged gestation, and long labor, with weak pains and operative deliveries are often encountered.

6. Postpartum hemorrhage is more common and may be a serious complication.

7. Irregularity of the fetal heart tones during pregnancy and labor is common.

8. Intrauterine fetal death before or in the early part of labor may occur.

9. When the heart tones are constantly irregular in the later weeks of pregnancy, cesarean section should be seriously considered.

10. Subsequent labors should be carefully observed for anomalies of the position of the fetus or the forces of labor.

THE PROPHYLAXIS OF POSTOPERATIVE PYELITIS

By Walter T. Dannreuther, M.D., F.A.C.S., New York

MODERN aseptic technic and expertly administered anesthesia have so reduced the hazards of elective gynecologic operations that the survival of the patient can no longer be accepted as the sole criterion of success. The real test of a pelvic surgeon's skill is an uneventful convalescence, with complete relief of preexisting symptoms. Careful preoperative study and preparation of the patient, particularly with reference to biochemical and functional disorders, have increased the percentage of symptomatic cures and contributed greatly to the lessening of postoperative morbidity. Yet, doubtless, all of us are distressed occasionally by the supervention of some annoying complication during an otherwise normal convalescence. It must be acknowledged that the most important preoperative responsibility of the gynecologist is to gauge the patient's vital resistance correctly. Profiting by experience, we soon realize that much can be done to prevent the mishaps which may arise during the postoperative period. A few cases of parotitis make one recognize the necessity for thorough preliminary inspection of the mouth and teeth, and oral hygiene; two or three cases of thrombophlebitis or pulmonary embolism are sufficient to impress one with the wisdom of encouraging the patient to move her extremities early and frequently after operation; the unexpected precipitation of acidosis or alkalosis in an apparently normal individual suggests the desirability of determining the preoperative values of the more important chemical elements of the blood; and all surgeons now appreciate that anemic and undernourished patients can be protected against the danger of postoperative shock by preliminary blood transfusions. In fact, the veil of obscurity has so lifted from the etiologic factors concerned in the causation of untoward postoperative sequelae that the time will come when the advent of any one of them after an elective gynecologic operation may be justly regarded as a reflection upon the operator's preliminary survey of the patient.

For several years I have made it a practice from time to time to review the results of my own pelvic operations, and in analyzing the five hundred consecutive cases immediately preceding December 31, 1926, I was chagrined to find that acute pyelitis had appeared six times during convalescence. This morbidity of 1.2 per cent is sufficiently high to merit attention, despite the fact that two of the most popular textbooks on the after-treatment of surgical patients fail to mention pyelitis as a postoperative complication. Technically speak-

TABLE I

Six Cases of Acute Pyelitis Occurring in 500 Consecutive Pelvic Operations
(Preoperative Data)

PATIENT	AGE	PREGNANCIES	URINARY SYMPTOMS	CYSTOSCOPIC EXAMINATION	CONSTIPATION	INDICANURIA	LEUCORRHEA	ENDOCERVICITIS
Case 1 Mrs. V. G.	30	0	0	Negative, including indigocarmine function test	+	+	+	0
Case 2 Mrs. F. P.	45	3	0	Extravesical pressure	+	+	+	+
Case 3 Mrs. M. M.	37	5	0	Negative	+	+	+	+
Case 4 Mrs. S. F.	28	2	Dysuria and frequency for 1 week	Trigonitis (Staphylococcus albus on culture of urine)	+	+	+	0
Case 5 Mrs. F. W.	28	2	0	Not made	+	+	0	0
Case 6 Mrs. R. S.	25	2	0	Negative	+	+	+	+

ing, some degree of pyelonephritis was probably present in these cases because pyelitis is usually secondary to infection of the renal parenchyma.

Although pyelitis occurs frequently in women and its predisposing causes are well known, the reason for its appearance as a postoperative complication at first glance seemed somewhat mysterious. Tabulation of the preoperative data (Table I) concerning the six patients involved disclosed that neither age, parity, nor preexisting urinary disturbances are of special significance. The etiologic importance of intestinal stasis, however, as manifested by constipation and indicanuria, is striking. Notwithstanding that five of the six patients had leucorrhea, only three suffered from endocervicitis, and in each instance the diseased area had been removed at the time of operation. Hence, in all fairness, I do not believe that the renal pelvic infection can be ascribed to a lymphogenous migration of microorganisms from the cervix. Furthermore, it is unlikely that the pyelitis was excited by ureteral reflux, as only one patient had a cystitis before operation. The diversified pathologic conditions and different kinds of operation (Table II) seem to exclude any particular type of pelvic disease as a factor of importance, so it is logical to assume that a long-standing intestinal putrefaction is the chief reason for the occurrence of pyelitis as a postoperative complication.

TABLE II

PATIENT	PATHOLOGIC CONDITION	OPERATION
Case 1		
Mrs. V. G.	Chronic recurrent appendicitis	Curettage
	Jackson's membrane	Appendectomy
	Right ovarian cyst	Oophorocystectomy
	Retroversion of uterus	Round ligament shortening
Case 2		
Mrs. F. P.	Lacerated cervix	Tracheloplasty
	Rectocele	Perineorrhaphy
	Parovarian cyst	Cystectomy
	Left broad ligament hematoma	Resection of broad ligament
	Retroflexion of uterus	Round ligament shortening
Case 3		
Mrs. M. M.	Lacerated cervix	Tracheloplasty
	Ectopic gestation (right)	Salpingo-oophorectomy
	Chronic salpingitis (left)	Appendectomy
Case 4		
Mrs. S. F.	Bilateral tuboovarian abscess	Salpingo-oophorectomy
Case 5		
Mrs. F. W.	Chronic metritis	Supravaginal hysterectomy
	Bilateral salpingo-oophoritis	
	Cystic ovaries	
Case 6		
Mrs. R. S.	Lacerated cervix	Tracheloplasty
	Rectocele	Perineorrhaphy
	Left parovarian cyst	Salpingo-oophorocystectomy
	Retroversion of uterus	Round ligament shortening
		Appendectomy

TABLE III

MANIFESTATIONS OF ACUTE POSTOPERATIVE PYELITIS

PATIENT	DAY OF ONSET	CHILL	TEMPERATURE	PULSE	NAUSEA	LUMBAR PAIN	HEADACHE	URINE CULTURE	ASSOCIATED CYSTITIS
Case 1 Mrs. V. G.	10	+	104.6	90	0	+	+	Colon bacilli	0
Case 2 Mrs. F. P.	18	+	103	88	+ (Vomiting)	0	0	Colon bacilli	0
Case 3 Mrs. M. M.	16	0	102.4	100	0	0	+	Colon bacilli	0
Case 4 Mrs. S. F.	14	0	102.2	100	+	+	0	Colon bacilli	0
Case 5 Mrs. F. W.	10	0	101.6	116	+	+	0	Colon bacilli	+
Case 6 Mrs. R. S.	10	0	100.6	86	+	+	+	Colon bacilli	0

Analyzing Table III, the following conclusions seem justified: acute pyelitis is likely to occur as a postoperative complication between the tenth and eighteenth days; the onset is usually sudden and is manifested by pyrexia, nausea, lumbar pain, and headache, although all these symptoms may not be present in any one case; the pulse may be rapid, but does not parallel the rise in temperature; patients with a high fever may have an initial chill, and the colon bacillus will almost invariably be discovered in the urine. In view of the fact that the clinical picture is not always the same, it is possible that some of these cases may pass unrecognized. The diagnosis is easily made by examining the urine microscopically and bacteriologically. Colon bacilli were found in the specimens from each of my six patients. The treatment consisted of daily colonic irrigations, forced fluids, hexamethylenamine and acid sodium phosphate, and a few irrigations of the affected renal pelves with silver nitrate after the acute symptoms had subsided. Prompt recovery followed.

Indican, or indoxyl potassium sulphate, is derived from indol, a product of intestinal putrefaction of albuminous substances. The indol is absorbed by the blood and oxidized in the tissues to indoxyl, which combines with potassium sulphate and is eliminated in the urine (Heitzmann). Small quantities of indican are present in normal urine, but since ethereal sulphates are products of decomposition, the intensity of an indicanuria varies in direct proportion to the amount of albuminous putrefaction in the small intestine. On these premises the occurrence of postoperative pyelitis can be justly attributed to a hematogenous colon bacillus infection, arising from an intestine harboring putrefying contents.

The corollary of this implies thorough intestinal cleansing and elimination of decomposing material before operation in all patients having excessive amounts of indican in the urine. Since reviewing the group of cases referred to, I have made it a practice to defer operation for at least two weeks in such patients, prescribing daily high colonic irrigations, Bacillus acidophilus cultures, large doses of mineral oil, a diet free from fats and sugars, forced fluids, and full doses of hexamethylenamine and sodium benzoate. Theoretically, hexylresorcinol would seem to be the ideal agent to protect the upper urinary tract from bacterial invasion, but the incidental necessity for restricting the ingestion of fluids to reduce the surface tension of the urine is a serious disadvantage in prospective operative patients.

CONCLUSIONS

1. Acute pyelitis occurred in six instances during convalescence in a series of five hundred consecutive patients subjected to pelvic operations; a morbidity of 1.2 per cent.

2. All six patients suffered from obstinate constipation and a pronounced indicanuria before operation.

3. An inexplicable rise of temperature between the tenth and eighteenth postopérative day, accompanied by nausea, lumbar pain, or headache should arouse suspicion of an acute pyelitis.

4. Colon bacilli will almost invariably be found in the urine.

5. Thorough preoperative intestinal cleansing and elimination of decomposing material, together with the administration of hexamethylenamine are efficient prophylactic measures.

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THE VOMITING OF PREGNANCY

BY EDWARD SPEIDEL, M.D., F.A.C.S., LOUISVILLE, KY.

THE incidence and mortality of eclampsia has been reduced enormously in localities and institutions where patients receive proper prenatal care. This is due to the fact that as soon as there is a rise in blood pressure and an albuminuria, strenuous efforts are made at once to combat these preeclamptic symptoms and ward off a possible eclampsia.

Unfortunately, the same zeal is not shown with the early toxemia of pregnancy. In spite of the fact that nausea and vomiting are always the forerunners of the more serious condition, hyperemesis, it receives scant or no attention.

From personal observation and the many articles in the literature of late, hyperemesis is now a more frequent condition than eclampsia. When we read the contributions of Titus on over 328 cases and of Harding on 200 cases, all serious enough to require the intravenous use of glucose, they report upon more patients with this condition than they would be able to present upon as preventable a disease as eclampsia.

As to the comparative seriousness of the two conditions, through recent improvements in therapy, eclampsia has lost a great deal of its terror with a distinctly lessened mortality. It is unfortunate, then, that by comparison no mortality statistics are available on hyperemesis gravidarum. An inquiry addressed to the Department of Vital Statistics received the following reply: "The data requested in your letter are not separately compiled in the Bureau of the Census, hyperemesis being included under international title 143 C (Accidents of Pregnancy). Abortion following vomiting of pregnancy is assigned to 143 A. Nephritis of pregnancy and vomiting of pregnancy are assigned to 148 C."

It is well known that deaths from hyperemesis are not reported directly as such. If they were, it would soon be evident to all of us that the condition causes as great a loss of life as eclampsia.

Even if it can be proved that the mortality from hyperemesis is negligible as compared to eclampsia, it still deserves consideration, because the distress and morbidity are greater than in eclampsia. In eclampsia there is a sudden onset with more or less unconsciousness and recovery or death generally within twenty-four to forty-eight hours. In hyperemesis, weeks and weeks of comparatively slight discomfort culminate in days of extreme distress accompanied by constant vomiting and even the inability to retain water. Any one who has ever passed through a severe attack of seasickness can appreciate the plight of the woman with hyperemesis, and it must be conceded that the condition is largely preventable. In many years of practice I remember no cases that originated among my own patients. Experience was only gained from consultation and referred cases. Upon inquiry the same holds good with other members of our obstetric staff. Only a few such cases have developed in our prenatal clinic since its organization. This no doubt is the experience of obstetricians and large institutions elsewhere, such cases coming to them for consultation or treatment in the final stages, with conclusive evidence of little or no care or attention in the early stage.

There is a very good reason for this. It is unfortunate for the pregnant woman that nausea and vomiting is classed as one of the presumptive signs of pregnancy. The physician recognizes it gladly as an additional aid in the absence of only one menstrual period, to an early diagnosis of pregnancy. To older women always on the alert in watching the newly married, it is the earliest telltale evidence of the condition which the new bride is so anxious to conceal. It is treated with a great deal of levity and is generally regarded as a necessary evil in such circumstances, so that little sympathy or comfort can be expected, until the condition becomes serious. The physician, with the understanding that the symptom occurs in 60 per cent of primiparas, is likely to be equally callous unless the situation becomes grave, yet it is in this early stage that treatment must be instituted to forestall a possible hyperemesis.

Of the many explanations for the condition, the rhythmical contractions of the uterus incited by the pregnancy may readily be responsible for the reflex irritation of the gastrointestinal tract that results in nausea and vomiting; a toxemia due to the presence of the fetus will naturally increase the irritability of the expanding uterus.

It must be realized that with the onset of pregnancy the uterus, a comparatively inactive organ, enters upon a state of intense metabolism. The intermittent muscular contractions that are at once instituted and that are later on recognized as Braxton Hicks' contrac-

tions control the circulation in the placental sinuses and cause a constant exchange of maternal and fetal products. One of the functions of the placenta is to store glycogen for the growing fetus until the fetal liver is able to store its own reserve carbohydrate, and during this period it is the glycogen of the maternal liver upon which it draws for its supply. At the end of the third month the fetal liver is fully formed and able to store its own glycogen, and then in ordinary circumstances the drain on the maternal liver should cease.

According to Harding this drain on the glycogen content of the maternal liver occurs with the very onset of pregnancy and the early nausea and vomiting is an indication of the resulting deficiency in that organ. The continued and increased demand for glycogen on the part of the rapidly growing fetus drains the glycogen content of the maternal liver; this deficiency causes nausea and vomiting, and the vomiting results in inanition and starvation with fatty infiltration and degeneration of the liver cells and ketonuria.

The sudden cessation of vomiting in many cases at about the third month of gestation is claimed to be due to the fact that the fetal liver has reached full development, and by storing its own glycogen relieves the maternal liver of the drain that is responsible for the vomiting. The neurotic element is explained as follows: Harding again states that a normal pregnancy is marked by heightened nerve reflexes. An unduly sensitive, sympathetic nervous system will result in the diminution of glycogen in the liver, because excitation of that system produces an excess of sugar in the blood at the expense of the glycogen in the liver; this naturally will be followed by nausea and vomiting. If the condition is largely neurotic, then suggestive therapeutics should control the vomiting. This will explain the sudden cessation of vomiting when the patient is removed to a hospital, away from the husband and the overly anxious members of her family.

The public is already educated up to the fact that albumin in the urine of the pregnant woman will lead to convulsions; they should now be taught that the early vomiting of pregnancy is a sign of equal gravity and that by treatment of that apparently simple condition the onset of the serious condition of hyperemesis can be largely prevented. Primiparae can be taught to put themselves in charge of their physician as soon as pregnancy is suspected and multiparae as soon as vomiting shows itself in pregnancy.

It is possible in all circumstances at least to make the patient more comfortable. The first essential for success is the prohibition of coitus. If the patient can sleep alone and even have a separate room, conditions are at once considerably improved. The woman should rest as much as possible, lying in bed longer in the morning when the nausea is generally aggravated. She should be relieved as much as possible from the duties of cooking. It is manifestly absurd to expect to relieve a woman of nausea and vomiting when she has to

prepare three meals a day. Even in the poorest families it will generally be possible to get the husband to prepare his own breakfast or get it elsewhere and add that much to bettering such a situation.

The old advice to send the young bride home on a visit to her mother has much to commend it. It undoubtedly eliminated the nostalgia which could readily be a factor with the young girl just torn from her home surroundings and suddenly initiated into the mysterious experiences of the marital state. It removed her from the embraces of the husband and the household duties incumbent upon her marriage. In such isolation there should be an immediate improvement, or the condition should respond readily to treatment. As to actual treatment, with proper arrangements made in her surroundings, the ordinary regulations of hygiene being observed, medication largely suggestive should relieve the condition. The intravenous administration of an ampoule of extract of corpus luteum daily seems to have a happy effect in this early stage, especially if the patient is compelled to come to the physician's office for its administration.

A one and a half grain dose of luminal-sodium three times daily, in addition, will have a tendency to allay the neurotic feature of the case and provide much needed rest.

In some instances the additional administration of ten drops of dilute hydrochloric acid in water before meals is necessary before success becomes assured. It is claimed that there is an hypochlorhydria in pregnancy conducive to nausea and that the administration of the dilute hydrochloric acid corrects that condition and should relieve the distress. If a laxative becomes necessary, tablespoon doses of milk of magnesia at bedtime will generally be effective and will alleviate the existing pyrosis. This with an enema of one-half gallon of warm water containing a tablespoonful of bicarbonate of soda, will empty the lower bowel and will to some extent relieve the acidosis. The feeding of patients in this early stage requires a little more thought and consideration than is generally given to it. Full meals are rarely desirable. Graham wafers, Holland rusks eaten dry are good foods to begin with. A dainty sandwich brought to the patient on a plate, lemonade, iced tea, and a few teaspoonfuls of pineapple juice in sweetened ice water will often tempt the patient and be retained. Salted pop corn will stay when nothing else can be retained and the dry cereals, puffed rice, corn flakes, etc., are in the same class. Peptonized foods are no longer administered per rectum as there is so little absorbed by that route, and the rectum soon becomes irritable and rejects it. More can be accomplished by reserving the rectal route for the absorption of needed sedative medication later on. It is always important, of course, to examine for any uterine abnormality, and if a retroversion is discovered, it should not only be corrected, but a supporting pessary should be inserted to keep the uterus

in its corrected position, the presence of the pessary also having a physic effect in relieving the condition.

Copeman's treatment of nausea and vomiting by finger or instrumental dilatation of the cervix has been recommended since time immemorial and is still practiced as evidenced by a case referred to me only recently. As early as 1884 Gill-Wylie warned against the procedure, unless it was conducted with antiseptic precautions, and he published the following directions: "After careful disinfection of the vulva and vagina with bichloride solution, the blades of the metal dilator dipped in pure carbolic acid, should be introduced into the cervix for about 1½ inches and slow dilatation practiced, followed by the application of glycerin gauze to the cervix." It is evident that with our advance in surgical technic even more care should be practiced, since interruption of the pregnancy may follow in consequence of the manipulations, and a serious infection result from a lack of such precautions. In a case recently referred to me a pyemic infection was inflicted upon a patient already in a low state of vitality from two weeks of incessant vomiting. In a primitive way relief from thirst and hunger in the graver cases can always be given to these patients, even in the poorest surroundings, by the gentle introduction into the rectum, with the patient lying on the left side, of four to eight ounces of water warmed to body temperature and containing two tablespoonfuls of honey or Karo syrup to the pint—an effective substitute for the glucose injections used in cases of hyperemesis.

When the condition does not yield to these simple measures, then nausea and vomiting takes on the aspect of hyperemesis, and extreme measures should be delayed no longer. There are certain well-defined and tried procedures in the treatment of this distressing condition that can safely be carried out even in the home, and these should be instituted without delay. Isolation in a hospital is, of course, most desirable, but if that cannot be secured, then conditions should be as closely simulated as possible in the private home, by removal of the patient to the brightest and sunniest room in the house and as far away from the kitchen as possible. The anxious husband and family and visitors should be excluded. The patient should be kept in a somnolent condition by the rectal introduction of 6 ounces of 10 per cent glucose solution, containing 60 grains of sodium bromide and 15 grains of chloral hydrate, three times a day more or less, to secure the desired result. One quart of decinormal saline solution should be instilled under the breasts daily to correct the dehydration and 500 c.c. of warmed 10 per cent glucose solution should be given intravenously daily until the vomiting is checked. No water or food or medication should be administered by mouth until this is accomplished. With the ingredients for normal saline solution put up in ampoules and 50

per cent solutions of glucose available in glass containers for dilution with fresh distilled water, such therapeutic procedures should be possible anywhere.

It is hardly justifiable, in view of the general advance in medical knowledge even by the laity, that extreme cases of hyperemesis with sordes on the teeth, dry red tongue, cracked lips, and bloody vomit should come to treatment. It should be classed as criminal if a pregnant woman is neglected to such an extent.

It is in extremely toxic cases, however, that therapeutic refinements must be added to the regular treatment to effect a cure. A hypodermic of pantopon with an ampoule of scopolamin is at times necessary in the initial treatment of hyperemesis to give the patient a temporary respite from the incessant retching. This combination will be found more agreeable than the morphine usually injected.

Glucose again, of course, is our sheet anchor in these grave cases, and it is fortunate that various avenues of administration are available. Oral administration is out of the question in this condition, and subdermal introduction is rather painful even with a little novocaine added to the solution. Fortunately the duodenal route can be used, and with the introduction of the tube sometimes preceded by spraying the throat with cocaine, glucose solution in definite amounts can be readily introduced into the system. The tube can be passed through the nose, if it is the intention to leave it in place a considerable time, and then water, glucose solution, other liquid foods, and even laxatives can be successfully introduced into the system.

The intravenous introduction of glucose solution is receiving most attention at present. Hendon presents an ingenious arrangement for the continuous administration of glucose solution—intravenous nutrition as he calls it. The median basilic vein is dissected up for about an inch and a half, and a specially devised blunt cannula with an obturator is introduced and tied into the vein beyond the first shoulder. The skin incision is then closed beyond the second shoulder, securing the apparatus safely in place. The glucose solution heated to proper temperature is put into a vacuum flask. Rubber tubing with a glass bulb arrangement for regulating the flow of the solution to the required number of drops per minute is connected with the tube; then at the lower end a glass nozzle with six inches of catheter tube connects with the cannula in the vein. The patient's arm is bandaged to a padded splint to prevent movement, especially flexion, and the glucose solution is allowed to flow. It can readily be estimated that with a flow of 20 drops a minute sixty fluid ounces of the solution can be introduced in twenty-four hours, and the apparatus has been kept going in one reported case for eight days. Hendon uses a 5 per cent solution but stronger solutions can, of course, be administered.

The intravenous administration of sodium bicarbonate with glucose is advocated by Wilson on the ground that hyperemesis is due to a disturbance of the carbohydrate metabolism. The ordinary percentage of carbon dioxide combining power or alkali reserve is 53 to 70. In pregnancy it will generally be found low, from 50 to 55 per cent. In consequence, the least disturbance of this slight balance will cause an acidosis.

As acidosis is caused by the abnormal formation of acid substances or the deficient elimination of acid substances normally found in the blood, the percentage estimate of the carbon dioxide combining power or the alkali reserve should give a desirable estimate of the individual metabolism.

A low carbon dioxide combining power of the blood is present in all cases of hyperemesis, and the percentage estimate should serve as an index of the severity of the condition. With an elevation of this index from its low level to normal or higher and a consequent relief of the existing acidosis by the glucose bicarbonate therapy, there should be a decided improvement in the condition. Continued treatment can then be rationally based upon the percentage estimate of the alkali reserve. Glucose will elevate the alkali reserve, but it can be raised more rapidly if sodium bicarbonate is given with it. Consequently, Wilson advocates the intravenous administration of 200 c.c. of 10 per cent glucose with 200 c.c. of 3 per cent sodium bicarbonate as the initial dose. The initial injection may be given upon the assumption that an acidosis is present in serious cases, but a second injection should not be given until the carbonate dioxide combining power is determined, in order to avoid an alkalosis. The solutions should be prepared and sterilized separately, heated to body temperature, and mixed. If heated together caramelization of some of the glucose may take place and render the solution unfit for use. There seems to be a rational basis for this treatment, and it should be given due consideration.

There is considerable controversy at present as to the intravenous administration of glucose with or without insulin in the treatment of this condition. Titus claims that the glucose infusion not only corrects the existing carbohydrate deficiency but restores the liver cells which have been destroyed by the toxemia and that the addition of insulin causes glycogen stores to become depleted by its demand for glucose to be oxidized and otherwise metabolized. In toxemia of pregnancy, therefore, in which storage in the liver and not combustion of the injected sugar is the desired result, the simultaneous administration of insulin is contraindicated. Titus admits, however, that insulin is indicated when the acidosis is so great that the patient is in coma.

Thalhimer claims that the rapid eradication of ketonuria and even of acidosis by combining insulin and glucose therapy seems to cause

relief of the nausea and vomiting in these nondiabetic patients. He claims that the use of insulin considerably shortens the treatment, as the vomiting ceases with the absence of ketonuria. As to any danger from the use of insulin in such cases, it is claimed that the amount used, that is, one unit of insulin for every three grams of glucose, can cause only sufficient carbohydrate utilization to hasten the disappearance of the acidosis. It would take much larger doses of insulin to cause loss of glycogen from the liver. Given as directed, the small amount of insulin cannot possibly prove an element of danger, as larger amounts are being given to nondiabetic children. Barbour reports the administration of insulin to forty nondiabetic malnourished children. One child, nine years of age, received 15 units daily for fifty-one days. With this point in dispute eliminated, the question then resolves itself into which is most desirable in the therapy of hyperemesis, storage of glycogen in the liver as advocated by Titus or rapid combustion of carbohydrates with insulin as advocated by Thalhimer. Thalhimer advocates the use of 1000 c.c. of 10 per cent or 2000 c.c. of 5 per cent glucose in very dehydrated patients, administered over a period of from four to five hours.

It may well be claimed that the intravenous administration of so large a volume of fluid will at times cause some cardiac embarrassment and requires the constant personal attendance of the physician during this entire period.

I had a patient complain of cardiac distress recently after only 150 c.c. of glucose solution had been introduced. The needle was at once withdrawn, and 500 c.c. of the solution were introduced the next day without discomfort.

In recent literature Clark reports two sudden deaths from acute dilatation of the heart, following intravenous introduction of 500 c.c. of properly prepared glucose solution.

Again, some patients show a considerable reaction followed by an elevation of temperature upon the introduction of the glucose solution. The solutions should always be prepared with pure glucose and freshly distilled water, and administered at body temperature. Various devices may be used to keep the solution at proper temperature during its administration. The solution properly warmed can be drawn from a vacuum bottle, an electric heating pad set at low can be fastened to the glass container holding the glucose solution, or the lower part of the tubing can lie on a hot water bag.

The strength of the glucose solution to be injected is another point of dispute. As just mentioned, Thalhimer advocates a 5 or 10 per cent solution; Titus, on the other hand, claims that 25 per cent solutions act more promptly and favorably than weaker solutions.

Wilder and Sansum have demonstrated that the average individual has a glucose saturation point, determined by the elimination of glu-

cose in the urine, after definite amounts have been administered intra-
venously. This saturation point has been placed at 0.85 grams per
kilogram hour for the average person. Taking the average woman in
the early months of pregnancy at 130 pounds, that would amount to
about 50 grams of glucose per hour for such a patient. In other
words it should take two hours to introduce 1000 c.c. of a 10 per cent
solution of glucose intravenously into such a patient. They also dem-
onstrated that a 16 to 18 per cent solution is the most desirable one
for introduction.

In our own hospital investigations with the intravenous injection
of glucose solutions of various strength, it was demonstrated that
glucose was invariably present in the urine if a solution stronger than
15 per cent was used.

Again, it has been found that when the normal tolerance of 0.85
grams per kilogram hour is exceeded either by too rapid introduction
or by the instillation of too concentrated a solution, that unconsumed
glucose at once appears in the urine and a profuse diuresis results.
As dehydration is such an evident feature of serious cases of hyper-
emesis, it can be realized at once that diuresis is not desirable at the
onset of treatment. Exactly the opposite condition prevails when
glucose is injected in eclampsia. The coma in that condition is admit-
tedly due to cerebral edema and with the intravenous introduction
of strong solutions, even up to 50 per cent, a profuse diuresis results
with absorption of this edema and a return of consciousness. Again,
it is evident that the degenerative changes that occur in the maternal
liver in hyperemesis also affect other organs, especially the heart, and
that it is dangerous in consequence to subject such patients to the risk
of a severe reaction or to cardiac distress following the introduction
of large quantities of fluid into the veins in a short time.

In extreme cases not responding readily to the glucose injections,
therapy by means of the duodenal tube will be found effective.

In a recent case, a primipara, thirty-eight years of age, with the first
conception after fifteen years of marriage, the duodenal tube was in-
troduced and kept in place for five days. After draining the gall
bladder, water, food, and medication were introduced through the
tube and retained, and when the tube was removed, oral feeding could
be resumed, and the patient carried the pregnancy safely to the end.

Polak advocates the transfusion of 300 c.c. of human blood by the
direct method with 500 c.c. of physiologic sodium chloride solution
added. It is well to remember this therapeutic measure in severe
cases that do not readily respond to the ordinary treatment and espe-
cially where for religious reasons an interruption of the pregnancy
will not be permitted.

Let us not delude ourselves, however, that all cases of hyperemesis
can be relieved by injections of glucose. Every now and then in a

serious case a patient that has ceased vomiting and is apparently taking an abundance of food will begin to show choreic symptoms and signs of polyneuritis. In such cases even the prompt evacuation of the uterus seems to be of no avail, as happened in a recent case in which that extreme expedient had to be used after the patient was apparently well over her vomiting and was taking an abundance of food.

Accordingly, it is suggested that severe cases of hyperemesis be subjected to intensive glucose therapy for four days. If marked improvement does not result then duodenal treatment should be added for four days. If at the end of that time decided improvement is not evident, then interruption of the pregnancy should be justifiable.

CONCLUSIONS

1. The next logical step in the reduction of maternal mortality and morbidity is in the prevention of hyperemesis.

2. Just as eclampsia has been largely eliminated by intensive treatment of the preeclamptic state, so by the prompt treatment of the ordinary vomiting of pregnancy, hyperemesis can be prevented.

3. The successful treatment of hyperemesis depends upon: absolute isolation of the patient from husband and family, which alone will often check the condition. Nerve sedatives, such as sodium bromide and chloral, per rectum. Dehydration combated by submammary introduction of decinormal saline solution. Inanition and acidosis counteracted by glucose intravenously. Interruption of the pregnancy if no prompt improvement and especially if choreic symptoms manifest themselves.

717 FRANCIS BUILDING.

THE TREATMENT OF VAGINISMUS

BY FRANCIS REDER, M.D., ST. LOUIS, MO.

REMEMBERING that vaginismus is sometimes an idiopathic, but generally a symptomatic disorder, the principal effort should be directed toward ascertaining the causal condition. This may require much care and tact on the part of the physician, and unless he can arrive at some definite conclusion as to the cause of the spastic manifestation, treatment can be only empiric and most likely of little avail.

The term "vaginismus" has been greatly abused and has served conveniently to characterize many of the conditions associated with pain about the orifice of the vulva. Vaginismus is an affection recognized by more or less active involuntary local sphincteric resistance at the ostium vaginae to penetration. There is an associated pain

which may be slight or severe. Diagnosis should offer no difficulty if
note be taken of the spasmodic contraction at the introitus. It is here
that ignorance or an insufficient examination is often culpable. Pain
or discomfort caused by coitus, no matter how severe, cannot be
designated as vaginismus. The term dyspareunia has been given to
this condition, meaning difficult or painful performance of the sexual
act. In this affection intromission is possible, whereas in a frank
vaginismus it is not.

The great difficulty encountered in vaginismus is to ascertain the
nature of the reflex spasm and, inasmuch as the condition can be
idiopathic or symptomatic, the solving of this problem may prove
perplexing.

The idiopathic type of vaginismus is a most unfortunate condition
and is due to an excessive nervous irritability, usually affecting the
whole system. It has been designated a hysterical anxiety neurosis,
not a neurosis based on either peripheral or central irritation. These
patients unconsciously develop the psychogenetic paroxysm and in the
majority of cases present the picture of a real hysteria. As long as
the irritable condition of the psychomotor areas of the sex organs
persists, nothing of an encouraging nature as to a cure of the existing
vaginismus can be expected. The treatment of this type of vaginismus
is a question for the patient herself; it is usually incurable.

In vaginismus of the symptomatic type we have a condition of a
more encouraging nature. Here the primary underlying cause is
usually some local anomaly. It may be an apparently insignificant
local disorder, or it may be a condition sufficiently serious to produce
much suffering. When a healthy woman with common sense com-
plains that intercourse hurts her so that she cannot submit to it, the
physician may at once make up his mind that there is some definite
cause for her condition.

By far the most common causes of the patient's sufferings in these
cases are fissures resulting from marital rents, or too frequent inter-
course, resulting in excoriations of the nymphae, or chronic vulvitis.
Furthermore, a tender myrtiform caruncle, an irritable hymen, a ure-
thral caruncle, an anal fissure, or a vascular degeneration of the
mucous membrane, usually on the inner surfaces of the nymphae, may
prove to be the cause of the total suspension of marital intercourse.

These are the pathologic lesions responsible for this distressing
condition, and upon their prompt recognition depends the future
happiness of the parties interested. It is by no means an easy task
to discover the exciting cause, during the first consultation, even upon
a most thorough examination and close inspection, and a subsequent
examination under anesthesia may become necessary. However, it is

advisable that the patient should first be examined without an anesthetic in order that the spastic condition about the introitus may be appreciated.

Additional reference is apropos to those cases of vaginismus not infrequently witnessed in the newly married. Although there is no ascertainable pathologic lesion about the ostium vaginae, a spastic condition of the parts is invariably produced when coitus is attempted. Upon what basis can this condition be explained? Usually the individual is of an emotional nature with the neuropsychic tendency predominating. She has read much and has heard more of the pain connected with defloration, which created in her the mental picture of fear, the most widely distributed of the emotions and the most powerful in its effect upon the organism. Harassed by this psychic trauma, it is only reasonable to assume that the fear of coitus, rather than the pain of contact, is the causative factor.

In an individual with a fairly stable neural balance, when a proper understanding as to the performance of the sexual act has been reached by both parties, the condition may disappear in a short time, either naturally or by the aid of simple measures. For these cases an extremely tactful and delicate consideration by the consultant, whenever his opinion is sought, will prove to be the most efficacious therapy.

In the more obstinate cases where there appears to be a somewhat pronounced neuropsychic personality, improvement may be accomplished by wisely chosen catharsis and by hydrotherapy. Opening the emunctories seems to open up mental paths favorable to a cure.

What is the prognosis of this affection? In those cases of vaginismus classed as idiopathic, where the individual is obsessed with a strong psychoneurologic element, the prognosis is not encouraging. The affection may remain indefinitely, becoming a permanent source of discomfort and woe. In those cases where the neural balance shows little deviation from the normal, and where the disturbance in the synaptic relations and in the cell chromatolysis has been slight, the outlook for a cure is very encouraging. The cases of vaginismus dependent upon anatomic causes, where a demonstrable lesion can be identified as the offending pathology, constitute the most favorable group for a cure. It is in this class of cases that a surgical measure is often deemed advisable to remedy the condition.

Usually every case of vaginismus is subjected to a course of treatment. This is as it should be. It may be possible that the application of a simple cerate, together with dilatation of the introitus, will relieve the condition. Again, the correction of any pathology present, such as fissure, vaginal or anal, the removal of a urethral caruncle, the incision of a resistant hymen, or the excision of oversensi-

tive carunculae myrtiformes, at the same time thoroughly stretching
the vaginal opening, may prove of great benefit to the patient and
eventually result in a cure.

There are, however, severe and obstinate forms of this affection that
are not much benefited by the foregoing procedures. The spas-
modic contraction and the hyperesthetic condition of the vulvar
orifice still renders the patient wholly unfit for her sexual functions.
For the relief of this condition it becomes imperative to resort to an
expedient that resolves itself into a surgical measure, the object of
which is twofold, that of severing some of the fibers of the muscles
directly implicated in the reflex spasm, and the enlarging of the vul-
val outlet. In performing this operation, an open procedure and not
a subcutaneous one, should be given preference.

There are two methods that are successfully practiced. In the one
an incision is made in the median line at the posterior part of the
vaginal orifice. This incision should pass three-fourths of an inch
into the perineum and extend one inch up the vagina. The cut mus-
cle should be allowed to gape, while the mucous membrane is dissected
free for a short distance on either side of the wound and sewed across
the gap. In the other, two lateral incisions on either side of the
fourchette converging in the median line just above the sphincter ani
to form a V are made. These incisions pass through the edges of the
constrictor cunni, and when carried deep enough also sever some of
the fibers of the anterior layer of the levator ani. The wound is
closed in a manner that will insure an enlarged vulval outlet. It is
advisable to use a nonabsorbable suture material. As soon as the
postoperative condition permits the introduction of a Sim's glass
vaginal dilator, it should be worn by the patient for two hours morn-
ing and evening for several weeks.

Of the two methods, my preference is for the lateral incisions.
Patients suffering with vaginismus are very irritable and become
quickly dissatisfied if the expected cure does not follow the operation
within reasonable time.

When should these stubborn cases be subjected to operation? No
definite rule can govern the time; however, it can be assumed, and this
assumption can be justly acted upon, when after four to six months
of proper treatment no appreciable improvement in the condition has
evidenced itself, and the patient no longer makes an effort to conceal
her dissatisfaction, it is my opinion that the time for operation has
arrived.

UNIVERSITY CLUB BUILDING.

MODERN OBSTETRICS IN THE HOME

By James R. Bloss, M.D., Huntington, W. Va.

THE invitation of your Secretary to present a paper before this honored Society brings me to the conclusion, that a plain practical paper would not be amiss, that it might bring forth a desired discussion of our everyday problems. My obstetric work is largely carried out in the homes of my patients, as is the custom in our section at the present time. Since one cannot change the scheme of things at will, I have made an honest effort to give to my clientele good care and conscientious service. It has constantly been my desire to improve the technic of obstetrics in home delivery that I might rob it of such dangers as may be possible.

It is not necessary to dwell upon statistics relating to morbidity or mortality among parturient women before this Association. Members of this body have been leaders in bringing these figures so forcibly to the attention of our profession. You deserve commendation for your efforts along this line, yet many questions are continually in one's mind as to the possibility of further reducing the number of fatalities and the morbidity in this particular field of work.

We must estimate the situation correctly. We must admit that at the present time by far the greater number of deliveries occur in the homes, and it will continue to be so for many years. That very desirable obstetric millennium when all pregnant women will go to our hospitals for confinement is in the dim and far distant future. If all were cared for in the aseptic delivery rooms of our hospitals by obstetricians, then the statistics would not be so disheartening.

The economic side must be considered, however, for not all patients are able to afford hospitalization unless they are to accept charity either from the hospital or from the physician. My personal experience is that by far the greater number of patients in moderate financial circumstances desire to pay for services rendered and prefer to remain at home and pay the physician for his care.

With the conditions as they are at present, it would seem that the wise thing to do is to study ways and means by which we may carry a better obstetric service to the very firing line, as it were. I speak feelingly of these things, for my work is done in one of the smaller cities of the country. Obstetric practice was not very highly regarded in this section until the past twelve or fifteen years. It was placed in the same category with anesthesia. Any physician and many of the neighbor-women were regarded as being thoroughly competent to render the service required. It is feared that too many physicians still regard this field of work in the same light. At the present time midwives do but very little work in this locality. Only two in fact

are so registered. Probably too many of our profession fail to appreciate the great advance of asepsis, even as applied to obstetric practice. Observation leads one to this conclusion in many instances.

Just here it is not amiss to mention a problem which, in my opinion, has a very great bearing upon the unfavorable statistical situation. The fact that we have established the value of asepsis and antisepsis leads to the performance of too many obstetric operations to hasten delivery that a little time may be saved. There are too many forceps deliveries; too many versions. Each has its particular place, it is true, but frequently they are injudiciously undertaken simply because it is felt that they are safe if one uses antiseptics. Many infants are sacrificed needlessly, and antiseptics do not always prevent infection. Additions to the therapeutic armamentarium of the obstetrician are not always free from danger in this same connection. The prevalent indiscriminate use of pituitrin falls in the same classification. When indicated and used judiciously in proper dosage, it is an agent of great value, yet untold injury has been done to mothers and infants with it. The performance of cesarean sections has become almost an epidemic. No reason can be offered to excuse this injudicious and pernicious rushing of so great a number of parturient women to the operating table. This widespread prevalence of needless obstetric surgery can only be emphatically condemned.

Every effort is made to give each patient thorough prenatal care. It has been and still is difficult to get women to appreciate the importance of this and to place themselves under the care of a physician at once after the passing of a menstrual period, that they may have an examination and be given careful advice. Gradually this is being overcome. The results cause patients to tell it to their friends, and they are encouraged to go to their own physicians under similar conditions. In this way prenatal care is being given more consideration, not only by the patients but by the physicians as well.

It does not prove to be a too difficult or time-consuming thing to make a thorough examination of our patient's general physical condition. First, is secured, as completely as is possible, a history—personal and family. The physical examination is carefully made, and always is included pelvimetry, external and internal, and an examination of the pelvic contents. Blood-pressure readings and urinalyses are made; a routine Wassermann test is made, and weight is recorded. It is insisted upon that the patient be seen every two weeks until the sixth week before estimated delivery and each week thereafter. At these visits blood-pressure readings are taken, and a urinalysis is made. General advice is given as to diet, exercise, and so on. Two to four weeks before confinement a list of articles to be sterilized is given to the patient. These articles are then brought to the office where the nurse properly prepares them. Sterilization is done at the

hospital, and the packages are returned to the patient with careful instructions not to in any way disturb the covers. These are to be left intact until opened by the nurse at the time preparations for delivery are being made.

At the onset of labor a visit to the patient is made. The examination at this time is usually limited to the abdomen. If it is found necessary to examine the condition of the cervix, the vaginal method is used. No vaginal examination is ever made without careful scrubbing of the perineum with soap and water, followed by Lysol solution. Sterile, dry, rubber gloves are always used, the hands, of course, having been previously carefully scrubbed. It is at this preliminary examination that I feel the greatest risk is taken. Personally, my obstetric conscience does not permit of less aseptic preparation than for an operation.

In the preparation for delivery, even in the most meagerly furnished homes, it has been possible to secure a satisfactory technic which does work out in practice. Naturally this necessitates carrying practically an operating room equipment to the home. A small portable copper sterilizer; such instruments as may be necessary for anything short of an abdominal section; sterile gauze, leggings, delivery sheets, sutures; solution for the infant's eyes, saline, mercurochrome, etc., are always packed and ready.

For a number of years it has been my practice to take a graduate nurse to each home confinement. In addition, when it is possible to do so, a senior pupil nurse accompanies us from one of the hospitals where the teaching of this branch is under my supervision. It is felt that in this way these young women can be shown that aseptic results are to be secured in surroundings most discouraging to the average graduate nurse, no matter how thoroughly trained, if she is inexperienced in home confinement. These student nurses are given a practical demonstration of home preparation.

For the past two years the mercurochrome preparation method has been used. It has proved to be eminently satisfactory and entirely practical. The nurse always gives an enema if the patient has not already had this, provided that the head is not engaged in the brim of the pelvis. The patient is then shaved carefully and washed with a solution of Lysol in boiled water. Mercurochrome is then applied over the abdomen from the umbilicus down; the perineum, the vulva, and the inner surface of the thighs to the knees are likewise painted with the same solution; one-half to one ounce of the mercurochrome solution is injected into the vagina, and lastly a sterile vulvar pad is applied. In case the labor is protracted, an additional half ounce is injected into the vagina at intervals of from two to four hours. During the period that this agent has been in use, no untoward effects have been noted. Four per cent solution is used both for the skin

and for injection into the vagina. One cannot but feel that if all physicians giving obstetric service in the home would adopt this method, the statistics so often quoted would at once show a decided improvement.

The patient once having been prepared, every effort is made and every precaution taken to avoid contamination. Sterile gown and gloves are always worn for these deliveries just as in the hospital. Naturally it is impossible to have the surroundings like those of a well-equipped institution. The effort is made to carry the principles of aseptic and practical antiseptic technic with us in this work and to apply them in actual practice. It is possible to do versions, instrumental deliveries, and immediate repairs (rather extensive in character) in the homes, as well as normal deliveries, and still secure satisfactory results. It has been a source of surprise and satisfaction to discover how well even extensive repairs progress, if these efforts to secure aseptic technic are constantly and conscientiously carried out.

In the management of labor in the home the same course is followed as is pursued in hospitals. At the onset a careful examination of the abdomen is made to determine the presentation and position; the relation of the head or breech, as the case may be, to the pelvic brim, and whether or not engagement has taken place. The fetal heart is located, and its rate and rhythm are noted. Experience in abdominal examination, after much practice, renders pelvic examinations, either rectal or vaginal, unnecessary in the great majority of cases, during the first stage of labor.

After satisfying myself as to the condition of the patient, I make it a rule to let her alone. The first lesson learned in my obstetric experience was a realization that the mechanism of labor was an unknown realm to me and that the understanding of this was of paramount importance, if one is really to become qualified to do satisfactory work. It was only after years of study and observation and experience that I was convinced that my place should be that of an observer on the side lines, so to speak, while nature was allowed the active rôle. The weak point, I think, in the practice of obstetrics, at least in the most of that done in homes, is due to the fact that the doctors did not understand the mechanism of labor when they were graduated, and have not grasped it since.

In the management of labor during any stage, but especially in the first, there is one thing to be discouraged. The patients should not be told to "bear down" or to make straining or expulsive efforts. My observation is that the only effect of this is an injurious one. It is more than a coincidence that we find the supports of the pelvic viscera so relaxed, and the bladder, uterus, and anterior rectal wall prolapsed to varying degrees. I am convinced that this so-called "bearing down" at an inopportune time is not only of no value, but in many instances is positively harmful.

Where conditions are progressing normally, the same course of watchful waiting is pursued during the second stage. I do not find that it is advisable in obstetric practice in the home to endeavor to assist nature by dilating the vaginal outlet. It is difficult enough to preserve asepsis, which is of prime importance, under such conditions, without undertaking the procedure of "ironing out" the perineum.

Experience and observation have shown me that the position of the patient's limbs is of importance during the completion of the delivery of the child after the presenting part begins to distend the vulvar orifice. I deliver practically all women in the dorsal position with the limbs extended upon the bed and the feet separated moderately. It is found that this causes an appreciable relaxation of the tissues about the vaginal outlet. In my experience flexing of the knees causes the structures of the perineum to be more tense and unyielding. Lacerations are not so frequently encountered and the necessity for episiotomy is not present to the same extent since this posture has been adopted. Anything which reduces the necessity for surgical procedures in home obstetrics has a very definite value. In my experience this position has proved valuable.

All lacerations are repaired immediately. It is felt that even slight first-degree lacerations of the vaginal mucous membrane, and of the skin about the vaginal orifice will provide a possible portal for infection. These slight tears are usually repaired before the delivery of the placenta and membranes. The bleeding is less, and this is to be considered where trained assistance is not available, as it seldom is in the home. If the lacerations are of the second degree, no repair is attempted until after the secundines are expelled. During this period the patient is allowed to react from the anesthetic, the nurse making preparation for the repair work.

One of the most difficult features of these repairs of a more severe nature is to keep the field reasonably free of blood, so that the structures may be approximated with a fair degree of accuracy. This is not an easy matter where no one is aseptically clean except the physician. It is my practice to administer a full cubic centimeter of pituitrin at the completion of the third stage. The subsequent contraction of the uterus, of course, lessens the difficulty of keeping the field fairly clear of blood. In repairs of cervical laceration the difficulties are often great. An expedient that I have found of great assistance when the perineal repair is begun is to place one or more large sterile scrub sponges, with a string or tape attached, in the vagina. Several packages of these sterile sponges are included in the list of supplies carried to each confinement.

In my repair work I always endeavor to bring the tissues together in separate layers, using interrupted sutures of either No. 1 or No. 2 chromic catgut. Personally this has proved far more satisfactory than an attempt to use a few deep through and through sutures which

include all of the tissues. It has been found that it is possible to secure satisfactory results in these repairs in the home with no assistance other than one nurse. The patient is turned across the bed with the hips brought to the side of it. A canvas leg holder with a loop for each leg is used to support the thighs, flexed to any degree desired. Sterile sheets protect the field and sterile sponges, instruments, and other needed supplies are at hand. The carrying out of this technic is not difficult if one has had some training and experience with it, and has been able to establish a definite routine of preparation.

After much study and trying out of various positions for forceps deliveries and the performance of versions in the type of obstetric service under consideration, a somewhat standardized procedure has been developed. Teachers of obstetrics from time immemorial have advised using the kitchen table, an unhinged door, etc. Personally I have never been able to discover kitchen tables which were satisfactory. They generally have one or more unreliable legs, are too high, too low, too short or too long. Doors do not have attachments which render them a suitable delivery table. In the face of these difficulties an effort is no longer made to find a substitute. The patient is turned across the bed and brought to the side until the sacrum rests on the edge. If two persons are available to support the limbs, they are instructed to sit on either side of the patient and each one supports a limb which is extended across his knees. The limbs, bed, and patient are protected by sterile sheets and towels. Where no individuals are available, it is found feasible to use two straight chairs with the backs turned toward the operator. The patient's limbs are extended, resting on the seats of the chairs, and the operative field protected with sterile drapes. The description may not be impressive; it can only be said that necessary operative procedures are carried out satisfactorily.

The management of the third stage is still one of assisting nature. It does not appeal to me that we should be in too great a hurry to deliver the secundines. After severing the cord between clamps, the uterus is watched for several minutes, one hand of either the nurse or the physician being placed upon the abdomen. The necessary attention is given to the infant; the cord clamp is applied, a sterile cord dressing adjusted after 1 per cent solution nitrate of silver has been carefully instilled in each eye and neutralized with normal salt solution. The child is then inspected for abnormalities. The nurse then proceeds with the cleaning and dressing of the baby, while the physician observes the mother's condition. If after an interval of thirty minutes the placenta and membranes have not been delivered, the uterus is massaged gently. In the majority of instances uterine contractions are so stimulated that the placenta will be expelled spontaneously. Rarely do we find it necessary to employ the Credé method.

Only in the rarest instances is manual delivery of the secundines necessary.

A postpartum hemorrhage at the beginning of my obstetric experience impressed itself so indelibly upon me that every precaution is taken to avoid a repetition of this. A full cubic centimeter of pituitrin is always given at the completion of the third stage, and a full dose of ergot by mouth, when the patient is able to swallow. Small doses of ergot are given to all patients who are delivered in the home at four-hour intervals for three or four days. It is felt that these procedures account, to some extent at least, for the absence of postpartum catastrophes, such as that mentioned above.

The question of anesthesia naturally presents itself. This is always dangerous ground upon which to advance in any meeting of medical men. Please bear in mind that the matter under discussion is that of obstetric service in the home of the patient, not in the delivery rooms of well-equipped institutions with skilled anesthetists available. The difference is unquestionably great. Much thought and study has been given to this phase of the work, and many different methods have been tried. In my own experience it has not proved satisfactory to attempt to employ rectal anesthesia in home confinements. There are many disadvantages to the use of ether in the home—open fires, evanescent effects, and so on. The use of nitrous oxide is not practical in the experiences I have had with it in this work. This leaves chloroform. In an experience of more than twenty years not one fatality has occurred to an obstetric patient from its use. It has been used carefully and with judgment. Morphine is given in the early stages when indicated. The dosage is usually small, and my experience is that its analgesic effects are enhanced if it is combined with small doses of hyoscine. Doses are repeated cautiously, depending entirely upon the progress of labor. Morphine in combination with magnesium sulphate has been tried. It has not proved so satisfactory, however, in my work as has the combination of morphine and hyoscine. It is found that if care is given to analgesia in the early stages of the labor, the completion of the delivery requires but little chloroform. In those cases where repairs are so extensive as to require anesthesia to a surgical degree, it is my practice to let the patient react while preparation for repair is being made; they are then reanesthetized, using ether. They do not stand a continuation of chloroform anesthesia well after the completion of labor.

I do not feel that one may neglect the after-care of these patients, if the best results are to be obtained. It is customary to continue daily visits for at least ten days, longer if necessary. A careful supervision of the after-care is exercised. This has been found necessary in order to prevent pernicious meddling by officious and superintelligent practical nurses and kind-hearted neighbor women who are helping out.

A case in point is in regard to giving parturient women intra-vaginal douches after the second day. It may surprise you to know that even in this enlightened day, in a city of 90,000 population, physicians still advise this. For some years much opprobrium was cast upon me because this time-honored procedure was so ruthlessly done away with.

The diet is advised. It is felt that the danger in home obstetric work is that of overfeeding for the first few days. Consequently one gets a reputation that he is too "cranky" about the food. Regardless, I continue the even tenor of my way, and in the end the results prove satisfactory to all parties concerned.

Patients are instructed as to the value of exercises in the bed after the third day, following out the method advocated by the Obstetrical Department of the Long Island College Hospital.

Finally, it is insisted upon that these women report to the office for an examination at the fourth or fifth week postpartum. In my opinion, we have not rendered our full measure of service until we are sure that their condition is one of normalcy.

At present the advisability of employing an additional nurse who will be put on an hourly nursing service to obstetric patients in their homes is under consideration. She would be expected to visit a certain number of patients each day, bathe the mother and child, take temperatures, advise the diet for the day, give enemas, etc. This idea has not been fully matured as yet. It can be seen where it would greatly enhance the service to those patiensts in moderate circumstances from an economic standpoint. Whether or not it can be accomplished without adding to their financial burdens remains to be seen.

These procedures from the first visit of the patient to the office to the time of their discharge are time-consuming and often tedious. Yet, if one is interested in securing the best end-results, they are more than worth while. Obstetricians of this type must of necessity be men enthusiastically in love with their work. Many patients cannot reward one financially to the fullest extent; they will pay to the limit of their ability, however, and it is personally found that the satisfaction of work earnestly and conscientiously performed more than makes up the deficit.

SUMMARY

1. The majority of confinements still occur in the home.

2. Pregnant women are certainly entitled to the very highest type of obstetric care.

3. It is possible to give this care in the home, if we earnestly strive to do so.

4. End-results prove that it can be accomplished in actual experience in a fairly large practice of this character.

418 ELEVENTH STREET.

Society Transactions

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOL-
OGISTS AND ABDOMINAL SURGEONS

FORTIETH ANNUAL MEETING

ASHEVILLE, N. C., SEPTEMBER 15, 16, 17, 1927

DR. DAVID W. TOVEY, New York City, read a paper entitled **Ladin's Sign of Early Pregnancy Compared With Hegar's.** (For original article see the current volume of the Transactions of the Association.)

DR. JOSEPH B. BACON, Macomb, Ill., read a paper entitled **A New Technic and a New Instrument for Operating on Rectal Stricture.** (For original article see the current volume of the Transactions of the Association.)

DR. DAVID HADDEN, Oakland, Cal., read a paper on **The Significance of Retrodisplacements of the Uterus and the Principles Involved in a Satisfactory Correction.** (For original article, see page 373.)

DISCUSSION

DR. WALTER T. DANNREUTHER, NEW YORK CITY.—It would be futile for one to attempt a discussion here of the comparative merits of the many operations that have been devised and advocated for the correction of retrodisplacements, but I believe that it is wise to place the stamp of disapproval again upon the Kelly suspension operation. Attending a clinic recently in another city, I was astonished to see the operator do two of these operations, despite the fact that I thought that most gynecologists had long ago been convinced that an intended suspension usually eventuates in either a fixation or a recurrence.

It is always necessary to differentiate cases of retroversion that are congenital from those that are acquired. A congenital uterine displacement produces no symptoms and requires no treatment. The symptoms, if any, are due to the incidental hypoplasia and not to the displacement, and any form of therapy directed to the correction of the displacement will result in disappointment. In cases of acquired retroversion, a distinction must be made between the so-called uncomplicated and the complicated ones. As a matter of fact, the retrodisplacements that are regarded as uncomplicated often give rise to symptoms because of the associated venous stasis in the perimetritic structures. This explains the insidious and delayed onset of symptoms so frequently observed.

A word regarding the pessary. I have removed pessaries from patients that have been inserted upside down, or back to the front, and in one instance both of these mistakes had been made. It is quite possible that the disrepute into which the pessary has fallen is due largely to the ignorance of some of those who apply it. A pessary cannot be expected to correct a retroversion. It is simply a device to hold the cervix back after the uterus has been replaced by the physician.

The various curves are designed for specific purposes, and if the principles upon which a pessary is moulded and constructed are thoroughly understood, this useful instrument will be misused less often.

DR. D. L. JACKSON, Boston, Mass.—In the operation for the correction of retroversion, we always have to consider the fascial shortening on the anterior wall of the vagina which holds the cervix forward. It is very simple to make a transverse slit at the bladder junction with the cervix, push the fascia upwards, and then sew longitudinally, thereby correcting that fault and allowing the cervix to drop back so that we do not get an anteflexion of the cervix when the retroversion is corrected.

DR. HERMAN E. HAYD, Buffalo, N. Y.—I agree with a great deal that all of the speakers have said, and perhaps disagree with some of them on some points. There is no question that we cannot do anything for a congenital retroversion. You may hitch it up, but the patient will suffer just the same. The classification that Dr. Dannreuther has made is very practical: those that have borne children and those that have not. Those who have no infection of tubes or ovaries will be cured by any kind of a round ligament operation. Those who have borne children have a disturbed pelvic outlet and that is the difficulty in connection with those cases.

Dr. Hadden seems to prefer the Webster operation. I have given up that operation altogether. I have given up all kinds of operations that involve the perforation of the broad ligament. In the first place, there will be adhesions, and the worst kind of adhesions follow that operation. Secondly, the incidence of a possible phlebitis is such as to contraindicate it. After all there is no operation that gives me as much satisfaction as the one done by the late Dr. Mann, where he simply loops the round ligament, and if the case is complicated by a fallen tube and ovary, it is a very simple matter to hitch up the ovary with a few stitches, and in doing so you will hitch up the tube. There is no question in my mind that a retroversion will sooner or later produce symptoms.

DR. GORDON K. DICKINSON, Jersey City.—There are two kinds of wisdom: one is the wisdom of the operating surgeon who has found a cure in a certain specific method; the other is the wisdom of the older man who has tried every method and failed in all of them.

My experience is that we are developing slowly along this line. Ten or fifteen years ago there were only one hundred methods of operating for retrodisplacements; now the doctor tells us there are one hundred and twenty. We are not centralizing on this because in our work we are not thinking, but are trying to follow a beaten path instead of working out each individual case and trying to treat it accordingly. We are all prone to forget that we have the patient as well as the condition. Some women have retrodisplacements and suffer, while others do not suffer, showing that there is a large neurotic element to be considered.

Dr. Dannreuther speaks of the venous congestion. It may be venous or capillary. I find that it is necessary to add to the operative procedure medical treatment. I have found apparently a specific effect in relieving the congestion by stimulation of the circulation in the pelvis by strophanthus. We must also add proper hygiene, vigorous exercise, and out of door life. The scrub woman and women in active life never complain of this condition. It is the sedentary, the fashionable woman, who has not enough exercise to throw the blood through the capillaries, who has trouble. We, as surgeons, should be hygienists and physicians as well, but there is no one type of operation in my experience which is going to give us 100 per cent results.

DR. JAMES E. DAVIS, Ann Arbor, Mich.—In considering the pathology of this condition, grossly one finds the uterus is enlarged, the endometrium congested,

the stroma hypertrophied or passed on to a condition of hyperplasia. The blood vessels have thickened. In other words, there is an early state of passive congestion. Later there is a condition of atrophy in the face of the condition of an enlarged and heavier uterus than normal. A cross-section of the wall will show that the tissue has changed and it is easy to observe with the naked eye that the walls are thickened and that the organ is not receiving a normal blood supply. Microscopically one will see that the nerve trunks have enlarged. Many uteri are removed by the operator because he has suspected myomas or fibromas, and when sectioned it is often a surprise to find only atrophy and fibrosis.

Reasoning from this picture of ultimate change, it is quite apparent that something should be done, a suspension, or some form of exercises following delivery, as J. O. Polak has advocated, or some other procedure to prevent this very obvious pathology. This can be a decision of importance in each individual case.

DR. HADDEN (closing).—I read the last two pages of my paper so hurriedly that I fear Dr. Hayd did not get my meaning. I severely criticized the Webster-Baldy operation, but what I do under favorable conditions is not the same operation. The Webster-Baldy operation can be improperly done in many ways and you see more failures from that than perhaps from the Gilliam, but it is when properly done, very effective.

DR. FRANCIS REDER, St. Louis, Mo., read a paper on **The Treatment of Vaginismus**. (For original article, see page 420.)

DISCUSSION

DR. LOUIS E. PHANEUF, BOSTON, MASS.—I want to emphasize the satisfactory method of operating by means of local anesthesia. I have operated upon two women recently, using morphine and scopolamine, and half of one per cent novocaine, injecting the mucocutaneous border and depositing about 5 c.c. of the solution in each levator. These women have consented to operation readily on finding that they would not be submitted to general anesthesia. I used a median incision, splitting in the middle line, and sewing laterally with perfectly satisfactory results. I have also delivered some of the women whom I had operated upon by this method.

DR. JAMES W. KENNEDY, PHILALELPHIA, PA.—During the past three years I have seen more cases of vaginismus than I have had during my entire previous experience. It is a real pathologic entity, and we have learned to be more considerate toward the patient than in former years, when we looked upon vaginismus as more or less of a neurotic condition, having little true surgical pathology. I recognize the condition as either a true vaginismus or a pseudovaginismus. If we recognize the pseudovaginismus as a symptom secondary to some local irritating conditions, such as urethral caruncle, inflammation about the meatus, painful fissures in the region of the vaginal orifice or the anus, rigid hymen, or that painful neuroma which may be a remnant of the hymen, then of course we have a happy solution of the annoying condition. I have seen a number of patients have a paroxysm of the muscles of the vaginal introitus if the examiner merely touched a skin tag which marked the remnants of the hymen, and I have also seen the same symptom emanate from simply palpating with the finger a tense nabothian cyst of the cervix.

Some of these patients are in need of sympathy and investigation from a nervous standpoint, others need surgery but they are all worthy of careful investigation.

DR. ADAM P. LEIGHTON, PORTLAND, ME.—This seems to be a malady from which the women in Maine suffer tremendously. It has been my peculiar experience to meet a number of them, and to follow them through to the divorce courts. It seems that many of these cases can be cured, and the penalty that follows married life may be avoided if we only intelligently try to treat these individuals.

One or two points have not been mentioned. Cases of recurring pyelitis have been the cause of vaginismus on several occasions, and cervical erosion with the resulting leucorrhea, which is sometimes overlooked, may be treated with a cautery and relieved. Another thing, a very small matter possibly, is the fact that many of these women have an alkaline urine, causing vaginitis and vulvitis and thereby irritating the hymen tags, which being changed to a normal acid reaction aids in a cure of the disease. The carunculae myrtiforme may be cut off, the Sims' glass dilator used and cocaine ointment applied. Where the urine has been changed to its normal acidity, good results have followed in many of these cases.

DR. HUGO O. PANTZER, INDIANAPOLIS, IND.—I wish to allude to one additional course in the treatment of these cases; namely, the mental influence of the patient over the condition. In some cases a curative effect may be obtained by having the patient practice relaxation of the perineal musculature, and doing it often during the day. It is surprising how many of these cases will respond under such practice.

DR. REDER closing).—Dr. Pantzer's suggestion about the psychic influence is a very good one, but the difficulty I have experienced is that almost anything may offend these women. All in all these are difficult cases to deal with.

DR. WILLIAM P. HEALY, New York City, read (by invitation) a paper on **Early Diagnosis of Cancer, Particularly From Gross Characteristics**. (For original article see page 353.)

DISCUSSION

DR. JAMES E. KING, BUFFALO, N. Y.—I believe that one of the frequent mistakes that the general practitioner makes in the diagnosis of cancer of the cervix is due to the teaching and belief that cancer of the cervix is essentially a disease of middle life. It has been my rather unpleasant experience to have found carcinoma in young patients and, without exception, they have been sent to me by physicians who confessed that failure to make the diagnosis was because the patient was under thirty. We should begin to correct this impression, for cancer is no respecter of age. In regard to the early tissue changes in cancer of the cervix, we must remember the various types of the proliferating and infiltrating growths, because of the difference in their prognosis and the ease in diagnosis. The proliferating type is, of course, very much less likely to extend early than is the infiltrating type; and, on the other hand, the proliferating type is much more easily discovered because it occurs on the cervical surface as an outgrowth.

The one type that occasions the most difficulty in diagnosis is the adenocarcinoma that develops in the canal of the cervix. I want to emphasize the fact that in suspicious cases the use of the sound is always of great help. Adenocarcinoma that develops in the canal can often be suspected where the sound in the canal produces bleeding, for it is seldom that a normal canal will bleed on the passage of a sound.

DR. HENRY SCHMITZ, CHICAGO, ILL.—We have adopted a very simple method of teaching the student the early diagnosis of carcinoma of the cervix as follows: The first stage is a nodule. A nodule is more often benign than it is malignant. If the nodule is incised and bleeds, it is probably malignant; if mucus exudes, it is probably benign. Hence, an immediate excision for diagnosis should be made. The second stage is an ulceration. The ulcer is usually small and has sharp, moth-eaten edges, and may be surrounded by an area of induration. If the ulcer is touched with a sound or a cotton applicator, bleeding will ensue. Free arterial bleeding probably means cancer. Hence a diagnostic excision should be done and a microscopic diagnosis made. If a carcinoma of the cervical canal or of the body of the uterus is suspected, then a sound should be inserted carefully and with

strict aseptic precautions. If bleeding ensues, and particularly if it continues for some time afterward, the supposition is that the patient has probably a carcinoma and a diagnostic curettage and microscopic examination of the scrapings should be made.

One should remember that carcinomas of the body of the uterus and of the cervical canal are very treacherous, as the external os may be perfectly intact. There are no characteristic symptoms of early carcinoma of the portio cervix or corpus. If bleeding occurs, we should suspect carcinoma until we can disprove it by microscopic evidence.

If the diagnosis of the early stages of cancer is taught, as Dr. Healy has suggested, and a woman will come to the doctor at about forty years of age for a health survey and repeat this at regular intervals, quite a number of early uterine cancers will be discovered, and of course, the good end-results of treatment will improve.

DR. W. S. BAINBRIDGE, NEW YORK CITY.—I was particularly impressed with the emphasis which Dr. Healy placed upon the importance of the clinical side in the diagnosis of cancer. It seems to me that the responsibility is with the clinician, and while he should not minimize the value of the pathologic tests, he should keep himself in close touch with all possible sources of information from a clinical standpoint. We do not always have the balanced judgment, but lean too much to one side or to the other.

I wish that I could feel that in our own country, as in Germany, the microscopic diagnosis is correct in 99 per cent of the cases, as reported by Dr. Strassmann. The percentage of accuracy in our laboratory tests is, I believe, much lower than this. Of course, the obvious case is not the difficult one: it is the borderline type. In one instance, I sent a set of slides for pathologic analysis to laboratories in Paris, Berlin, Rome, and to eight of the leading laboratories of this country, and I received seven different diagnoses of the one pathologic specimen, in return. These included diagnoses of sarcoma, carcinoma of the various types, tuberculosis, syphilis, and Hodgkin's disease. I may add that a succinct record of the clinical aspects of the case accompanied the set of slides.

There are one or two points where Dr. Healy may not have made himself clear. I do not think that he meant to say that one out of every ten human beings living today will die of cancer. Even the most extreme statistics do not present such figures. The accepted records show that of those over thirty-five years of age one out of every seven women will develop cancer, or die of cancer, or die with cancer. If we include all children and persons under thirty-five years of age, we cannot say that one out of ten of the human family will die of cancer.

Finally, the investigation of the Metropolitan Life Insurance Company of 125,000 deaths from cancer among one million industrial policy holders, concludes that "the actual increase in the death rate is small * * * much smaller than might be inferred from analysis of published death rates."

DR. JAMES E. DAVIS, ANN ARBOR, MICH.—Frequently in our routine practical work we proceed at times in a hysterical way to the diagnosis of cancer. When a case is presented to the surgeon, physician, or pathologist, a very prompt and hasty diagnosis is expected. This is very difficult in many instances when you consider all of the factors involved. In the far-advanced cases one is dealing, of course, with a very easy problem, but in the cases that are being discussed today, diagnosis is difficult. One is called into the operating room from time to time and asked to pass upon certain tissues by instant ocular examinations. A more adequate opportunity should be provided by assembling of the complete data and a more extended examination permitted.

Dr. Healy has quoted Maude Sly in regard to the value of heredity, and also as to the significance of irritations. It takes a little time to develop the history

of old and forgotten irritations in the area of newgrowth. I believe we are not in a position to diagnose these very difficult cases until we have the data all before us. The only logical and safe way of proceeding is by the eliminative method. I am in the habit of teaching my students that the whole field of pathology can be encompassed by making a few simple divisions. First, consider malformations or developmental defects; second, consider vascular conditions; third, inflammatory conditions, and fourth, whether it is a degenerative or a newgrowth change of benign or malignant type.

If one will leave the cancer decision until the last it may be easy to rule out a malformation or a developmental defect. Then proceed to the other conditions, and one will find the greatest trouble with the inflammatory changes which include the other types of changes. The most frequent decisions will need to be made between the inflammatory and newgrowth changes.

DR. F. S. WETHERELL, Syracuse, N. Y.—I would like to ask what Dr. Healy's view is in regard to treatment of early carcinoma of the cervix; whether he is using radium in all cases or whether in some of the very early cases he resorts to hysterectomy? I would particularly like to know what his experience is with adenocarcinoma of the cervix. Some men working in this field say we may treat squamous-celled carcinoma with radium, but that adenocarcinoma is resistant to the gamma rays.

DR. HEALY (closing).—Replying to Dr. Bainbridge, I probably left out the word "Adults." I meant to say that one of every ten adults will die of cancer, according to the statistics we handled, and one in every five women between the ages of forty-five and sixty-five. That is all right if you have a gambling instinct. You can say you have a ten to one chance anyway and need not worry about it. But those are facts.

We are endeavoring to discuss diagnosis at the time when cancer is, in our opinion, curable and that of course is not in the textbooks.

In regard to the question of adenocarcinoma of the cervix, those cases that develop in the canal usually are infiltrating and are advanced when they come under observation. They are not any better or any worse than the infiltrating epidermoid carcinomas. If in a very favorable case of cervical cancer you see fit to do a Wertheim operation, you may do so, but we feel that the patient will do perfectly well without that operation and without the risk of mortality if she is treated with radium, either with or without x-ray. We combine x-ray with it. Some very excellent men use radium entirely. We believe irradiation in the early stages will give better results in the sum total of cases, without morbidity and mortality, than surgery.

Address, on **Lawson Tait and His Contributions to Abdominal Surgery,** by Dr. John Harvey Kellogg, Battle Creek, Michigan. (See the current volume of Transactions of this Association for this address.)

Dr. P. Brooke Bland, Philadelphia, Pa., read a paper entitled **Hydatidiform Mole—Is the Expectant Plan of Treatment Justified?** (For original article see page 390.)

DISCUSSION

DR. PALMER FINDLEY, Omaha, Neb.—I take it that Dr. Bland is of the conviction that a mole can be judged as to its malignancy by a microscopic examination, and I am aware that he has abundant support in the position he assumes. Neumann states that the chorionic epithelium suggests malignancy; Mutz thinks

that the deep invasion of the musculature suggests malignancy. These and other authorities suggest that we have a reliable means of judging whether a mole is malignant.

Recently a splendid article has come from Hitchmann in which he says we have no reliable guide as to the malignancy or nonmalignancy of these moles.

I was first interested in the subject in 1903 when I searched the literature, going back as early as the sixth century. I could find only 210 cases—a most insignificant number as compared with the number that must have occurred during that long period. There was 25 per cent mortality, 16 per cent of which was from malignancy. I do not believe that that was an expression of actual facts. Most of the cases were recorded because there was some eventuality of special interest, sepsis or malignancy or what not.

I wish Dr. Bland would try to gather some evidence as to the frequency of hydadidiform mole. We hear that it is as high as 70 per cent in the early interruption of pregnancy and that it is exceedingly frequent in normal deliveries, even as high as 30 per cent of the placentas showing some myxomatous changes.

I would like to make this suggestion, as I did in 1903, that if two weeks after a mole is delivered there is found in the serapings actively proliferating epithelium, a hysterectomy be done.

If there is no evidence of malignancy at this time, there would likely be no malignant development in the future.

DR. JAMES E. DAVIS, ANN ARBOR, MICH.—The work of Dr. Blair Bell, in England, in the lead treatment of cancer received its greatest impetus in this worker's mind after he had studied the biology of the placenta.

It is most interesting that in this condition we have both a degenerative process and a newgrowth occurring perhaps at the same time, or at least the newgrowth process follows quickly after the degenerative process.

The mesothelial stroma of the villus begins to degenerate and is the first sign we can recognize to enable diagnosis of this condition. This degeneration in the central part of the mesothelial stroma will progress until the entire stroma has been destroyed. Then the activity of the border epithelial cells appears to begin in earnest, and an actual secretion is thrown out sufficient to fill the cavity left by the degenerative process.

In regard to the change from the benign condition to the malignant. It is often difficult to differentiate these two conditions. However, the same criteria used in diagnosing difficult cases of malignancy should be applied here. It is the differentiation of the cells—their departure in this differentiation process from the normal. The normal type of epithelial cell in the chorionic villus may grow and differentiate, and there is just a little departure from normal. That departure increases greatly as the invasion takes place into the uterine wall, and it is upon that change we must rely most for our differential diagnosis of the condition.

DR. BLAND (closing).—The clinical records at the Jefferson and St. Joseph's Hospitals disclose ten cases which had been recorded during the last few years. Six of the patients developed chorionepithelioma; four recovered and two died. Four of the patients had simple benign moles. Two recovered and two died of hemorrhage.

DR. HENRY SCHMITZ, Chicago, Ill., read a paper on **The Etiology and Treatment of the Bleeding Uterus**. (For original article see page 344.)

DISCUSSION

PROF. STRASSMANN, BERLIN.—I agree with all that Dr. Schmitz has told us. It is interesting especially to see the difference in the cases of bleeding uterus that occur in the years from fourteen to nineteen or twenty and those which occur at

about fifty years of age. In some of those cases nothing but conservative treatment really helped. The chlorosis of young girls has almost disappeared for some years, perhaps because no compressing of the organs by a corset now exists. The number of cases of bleeding uterus has increased very much. Everything possible should be done to build up the constitution. We give endocrine remedies, but they do not often have much effect. Dr. Thaler, of Vienna, proposed to make an incision, take off a part of the ovary, and sew it together. Personally, I do not like to use x-rays in such young patients. We now make irradiation of the spleen, and sometimes that is of benefit.

I remember one patient who had had this condition until her twenty-seventh year. Curettement was performed several times and all conservative methods of treatment exhausted. She had a child but developed afterward the same condition. We could find nothing in the uterus or ovaries to account for it. When we performed extirpation of the uterus, because nothing else would help her, we found sclerosis of the uterine arteries. There will be a certain number of cases, where you cannot help the patient in any other way than by operation.

DR. WM. P. HEALY, New York City.—I assume from the way Dr. Schmitz handled the topic that he was trying to avoid as much as possible the question of malignant disease and that he was considering benign conditions. Naturally it divides itself at once into the intrinsic uterine conditions and those that are outside the uterus. Our attitude will vary, depending upon the patient's age. When a young girl appears with symptoms of menorrhagia, we decide we must be dealing with a condition of endocrine imbalance. Occasionally it will be a combination of imbalance plus displacement.

I feel as Dr. Schmitz does, that we should avoid the use of radium in these young women. I think curettage is justified when other treatment fails. As a matter of fact, when these girls of fifteen or sixteen are sent to us at the Memorial Hospital, we never curette them. We put them on endocrine treatment and keep them in bed. We almost invariably correct the situation along medical lines. However, occasionally we have a patient who will not respond to medical treatment or curettage, and we then give them a small intrauterine dose of radium.

DR. PERCY W. TOOMBS and DR. I. D. MICHELSON, Memphis, Tenn., presented a paper on **Clostridium Welchii Septicemia Complicating Prolonged Labor Due to Obstructing Myoma of Uterus. Report of Case**. (For original article see page 379.)

DISCUSSION

DR. JAMES E. DAVIS, Ann Arbor, Mich.—These types of infections may be carried in from without by the operator or attendant. The most common source is from the intestinal tract. The Clostridium welchii may be found in the lower intestinal tract of most individuals. This leads to important clinical considerations. Why is it that we have such a serious condition to deal with in the cases of Clostridium welchii infection? The only rational explanation is that many of the organisms do not produce a toxin. There are some strains that evidently produce very dangerous toxins, and it is in these cases that serious results usually follow.

From the clinical or operative standpoint, a very practical observation may be made; namely, that the symptomatology of stasis that carries on to the condition called ileus, or to peritonitis, if compared with known cases of infection from the Clostridium welchii, will impress one with their almost exact similarity. It is usually very discouraging to meet in surgical practice a case of ileus or stasis because it is progressive.

The essayist in his classification has referred to two groups: the group where abortion has occurred, and the nonaborted group. In the latter group he has

spoken of obstruction in practically every division he has made. So in a practical way it is very important to remember that these three conditions may be identical.

The standardized treatment of ileus is drainage. Adequate drainage has been the best treatment up to the time that antiserum has been used, and in the results derived from the antiserum treatment, it has been observed that just as soon as the bowel begins to move normally, or continues to move normally, the patient usually commences to improve. It would be rational treatment to begin the serum whenever there is ileus or where there is a progressive stasis, or a peritonitis with marked progressive stasis. (See report of experimental work in the Brit. Med. Jour., Vol. XIV, October, 1926.)

DR. L. A. CALKINS, UNIVERSITY, VA.—We have had two cases within the last year and a half. I saw each of them about forty-eight hours after the first incidence of increase in temperature. In neither was the diagnosis made until the time of delivery and the presentation was normal in both cases.

The first case was that of a multipara whose membranes ruptured early. The fetus in this case was extremely edematous. Pure cultures were obtained from the tissues of the body of the child and from the blood of the mother. This woman died six hours after delivery.

The second patient was admitted to the hospital, six months pregnant. We tried to get some confession of interference. The physician had not examined her. She came in with a temperature of 103°, pulse 130, membranes unruptured, slight labor pains, and no bleeding. We did not suspect gas-bacillus infection until she miscarried, about thirty hours later. The membranes ruptured, and the amniotic fluid was frothy. In three instances pure cultures of gas bacillus were obtained from the blood. This woman was immediately put on large doses of antitoxin. Within twelve hours this patient's temperature became normal or subnormal and remained so for a week. Her hemoglobin had dropped from 65 at the time of delivery, with almost no blood loss, to 40 the following day. Forty-eight hours after delivery the hemoglobin was 30, and in the next twenty-four hours it was 23. It took us about forty-eight hours in her case to obtain donors. We then started transfusions, and the hemoglobin not only came up but stayed up after the transfusions.

Coincident with the rapid decrease in hemoglobin, there was an appearance of marked jaundice. This also cleared up following the transfusions. This patient has remained alive now some time over two weeks following delivery. Another clinical feature was variation of the leucocyte count with no apparent rhyme or reason for the increase and decrease. Four days following delivery she developed complete anuria. She passed in the next forty-eight hours less than 50 c.c. of urine; then gradually the kidneys began to secrete and the day before yesterday she passed 3000 c.c. of urine, yesterday 4500 c.c. (Subsequent Note.—Blood urea rose to 84 and the patient died of uremia. Postmortem revealed moderate pericarditis as only focus of infection.) She had not had at any time a typical bluish discoloration of the skin.

DR. FREDERICK H. FALLS, Chicago, Ill., read a paper entitled **A Study of Pregnancy and Parturition in Primiparae with Bicornuate Uteri.** (For original article see page 399.)

DISCUSSION

PROFESSOR P. STRASSMANN, BERLIN.—This paper was one of the reasons, why I made a journey to your country.

We have heard about the bicornuate uteri and the dangers to the mother and baby. It is very necessary to have an operation that will give us living mothers

and living babies also. Here nature has forgotten to unite the two halves of the womb, therefore, these patients have dysmenorrhea, abortion, premature labor, placenta previa, breech presentations, obstructed labor, and puerperal fever. They have also chronic inflammations of the appendages and wrong diagnosis, or useless operations. They are now operated upon by uniting the two halves into one cavity.

For the uterus bifidus, the so-called double uterus—I proposed a new operation. We have to differentiate between the uterus didelphys (bicornis and the uterus bilocularis (septus) with or without double vagina. The bicornuate uterus must be incised from one horn over the saddle to the other horn. Then we unite the right and the left halves with deep and superficial catgut sutures in a similar manner as in cesarean section. Altogether I have operated on 22 patients. I operated thus even in a case of hematocolpos and hematometra of the right side. At first I opened the closed right vagina; the blood escaped, and the hematometra was emptied. The next menstruation was painful; a tumor still remained. I had to remove the right tube for hematosalpinx. In that second operation I reunited the two halves of the uterus. This patient has since the operation been delivered of a baby out of the reunited uterus. The new uterus does in those cases not rupture; it holds fast.

I have also resected the uterus in the earlier months of abortion. One of my patients was bleeding; she wanted to have a baby; but the uterus could not safely be emptied in the usual way. Abortion had to be finished by scraping the right half of the womb after colpotomy and opening the fundus. I reunited the uteri by the same operation (vaginally), but she has not yet had a child.

In other operations I had to deal also with the chronic inflamed appendages.

In infected cases, where there is already inflammation of the appendages, the patients will return to normal condition, when they get a "united uterus."

The uterus septus is treated by cutting the septum and closing the cavity without any loss of substance, just as cutting the vagina septa. Afterwards there will be normal conditions for confinement.

DR. WALTER T. DANNREUTHER, New York City.—I have had 13 cases of malformation in twenty-one years, and one of the most important features of Dr. Fall's presentation, from the teaching standpoint, is that it controverts the legendary ideas that these patients are usually sterile. Eleven of my patients were married, and six of them produced collectively 16 children and 11 miscarriages, and 8 of the miscarriages were induced. So it is obvious that the incidence of pregnancy is as high in these women as in others.

Dr. Fall was fortunate that the uterus in his first patient was of the septate type. It is sometimes difficult to distinguish the septate from the bicornate uterus during pregnancy, and whereas a rupture of the "dividing membrane" might be comparatively safe in the septate anomoly, it might also be extremely dangerous in the bicornate uterus.

The high position of the bladder in cases of bicornuate uteri is to be expected, because the peritoneal reflection of the bladder is carried up into the sulcus between the corpora and fuses with the peritoneum of the culdesac, thus forming the rectovesical ligament. In operating on these patients, as I have had occasion to do for fibroids several times, it is always wise and usually essential to divide this rectovesical ligament well back on the cleft between the two uterine bodies, to free the upper limit of the bladder. The generous transverse incision across the uterovesical peritoneal fold that is customary in the technic of an ordinary hysterectomy would jeopardize the bladder wall, hence, the necessity of the preliminary detachment on the posterior surface of the sulcus.

DR. LOUIS E. PHANEUF, Boston, Mass.—My experience has been limited to three cases. I had one case that I followed through five pregnancies, three pregnancies in the right horn and two in the left. Unfortunately, I was not able

to obtain a living child. The longest pregnancy was for six months. I operated upon this patient after a second pregnancy, dividing the vaginal septum and removing a myoma at the junction of the two horns.

DR. FALLS (closing).—Concerning the remarks of Dr. Dannreuther regarding the difference between the septate and bicornuate uterus, I wish to say that bicornuate uterus is a broad term which embraces several varieties. The most extreme degree of bicornuate uterus is the uterus didelphys. The uterus arcuatus is the simplest form. The uterus septus and the uterus subseptus are variations between these two extremes.

Regarding the discussion of Professor Strassmann and his operatoin on the true bicornuate uterus. This operation implies a true separation of the two horns of the uterus. Uteri of this type have given such distortion and are so striking clinically, that numerous reports have been made in the literature. On the other hand, we could find nothing concerning labor occurring in the milder types of bicornuate uterus, such as the uterus arcuatus and uterus septus. It is logical, however, to expect that the milder degree of uterine deformity would be more common than the extreme degrees, and it is probable that because complications are less constant and striking that this condition has been overlooked. When an oblique or transverse presentation occurs in a primiparous woman, it is well to consider the possibility of one of the milder forms of bicornuate uterus. Such patients must be watched more carefully than those with a normal pear-shaped uterus. My first experience with this type of case surprised me considerably. I recognized that something was wrong because of the position of the head and the rapidity of the heart tones. I was struck by the fact that two babies died in this type of uterus without other discernible cause. On searching the literature I found nothing covering this particular type of case. Since we have been observing these cases, members of my dispensary staff send in patients with a notation regarding this deformity. Almost invariably labor in these women will show some abnormality, whether it be a postpartum hemorrhage, version, or extraction, a necessity for forceps or manual removal of the placenta, or some other complication.

DR. EDWARD SPEIDEL, Louisville, Ky., read a paper on **The Vomiting of Pregnancy**. (For original article see page 411.)

DR. PAUL TITUS (reader), DR. PAUL DODDS, and DR. E. W. WILLETTS, Pittsburgh, Pa., presented a paper on **The Fluctuation of Blood Sugar During Eclampsia, and Its Relation to the Convulsions**. (For original article see page 303.)

DISCUSSION

DR. F. S. KELLOGG, Boston, Mass.—Regarding Dr. Speidel's paper. During the past three years we have followed in the hospital a routine treatment of vomiting of pregnancy with marked success.* With this treatment we have done many less therapeutic abortions than before for this condition. We have as a routine given forced hourly feeding, forced fluids, large amounts of bromides, and isolation; stomach washings when the patient persisted in vomiting in spite of these measures. and last but not least, the removal of the vomiting pan from the room so that if the patient vomits she must do so on the floor or on the edge of the bed.

Forced feeding consists of giving the patient water, milk, and malted milk alternately every hour from 7 A.M. to 9 P.M., and one quart of glucose solution (5 per cent) every four hours by rectum. The glucose instead of being given as before in tap water, is given in slightly hypertonic or normal saline solution, for the reason that a careful study of the stomach contents has shown that there is no

free hydrochloric acid in the stomach in these cases. One quart of salt solution every four hours by rectum alternating with the glucose is used as well from 7 A.M. to 9 P.M. Salt and glucose under the skin and occasionally intravenously are used as needed if the rectum rebels. From 6000 to 7000 c.c. of fluid—glucose, salt solution, water, and food are used in a day. Thirty grains of bromides are given in each alternate tap. The nights are left free from treatment. If the patient vomits persistently after treatment is well established, the stomach is washed after each vomiting. The response to that treatment has been at least 300 or 400 per cent better than our results previously under any modification of this régime. It is a very rare thing now for us to do a therapeutic abortion, although we get some patients in very poor condition.

One more detail which we have found valuable is that we do not starve the patients for twenty-four hours as we did, because so many patients have been thrown from a mild degree of acidosis into a severe acidosis by this starvation period. We start feeding immediately.

Regarding Dr. Titus' work concerning blood sugar in eclampsia, it is a most stimulating and productive thing. Technically, it is very difficult to do, and he and his associates are, I think, entitled to great credit. The investigation shows that this blood-sugar phenomenon undoubtedly exists in eclampsia, that all the work that has been done on blood chemistry in eclampsia needs to be reconciled and checked, although we should be very guarded in drawing sweeping conclusions from it at the present moment and in attributing the convulsions to the low blood sugar necessarily.

DR. GEORGE W. KOSMAK, NEW YORK CITY.—In taking up Dr. Speidel's paper there is very little to be added as he has summarized in an exceedingly complete fashion all that we know of the modern treatment of the vomiting of pregnancy. I fail to note, however, that he paid sufficient attention to some of the reflex causes of vomiting. For a time we were led to disregard many of these reflex causes and direct more of our attention to methods of treatment that were apparently based on laboratory studies. I am firmly convinced, however, especially basing this on observations of recent years, that the condition of the cervix is a very important factor, particularly in the younger women. I believe it is quite necessary in the very early case, where vomiting is a more or less prominent symptom, to make a visual inspection of the cervix and to correct the erosions that we so often find. We are accustomed to look for these things in older women, and yet I have been surprised in recent years to find that a large number of young pregnant women with local lesions of the cervix, who vomited considerably, have improved when the lesions were corrected.

Another cause for serious vomiting, which is often overlooked is gastric ulcer, and this is particularly noted in young women who present a very anemic appearance and in whom vomiting of blood constitutes one of the symptoms.

One more important point is that abortion in these girls should not be long delayed. Too often in the past we have tried everything and then found that abortion threw the balance in the wrong direction. Abortion should always be considered, especially in women of the highly nervous type. I have seen a number of cases of mania where abortion was not done until it was too late to accomplish the desired relief.

Dr. Titus' paper is one of the most convincing arguments for a line of definite, specific treatment in the toxemias of pregnancy that I have ever listened to. Whether others will show the same thing in checking up his results remains to be seen. I am sure if the possible pitfalls are noted, to which he has called attention, probably the same facts will be demonstrated by others. It seems quite conclusive that the peculiar drop in blood sugar which antedates the convulsions is actually present, as he has so very well shown in his observations.

May I refer to the overdose of glucose. I see no objection to giving an overdose because the sugar is immediately thrown off in the urine and there is nothing to fear from that. This should controvert the claims of those who advise insulin in these cases.

DR. G. D. ROYSTON, St. Louis, Mo.—In connection with Dr. Speidel's paper, I want to call to your attention that there are three progressive stages in the vomiting of pregnancy: anorexia, nausea, and vomiting. The time for treatment is most indicated during the first two stages, and these patients should be treated actively, immediately, and vigorously. We give much larger doses of glucose and saline solution than Dr. Speidel mentioned, usually from 500 to 1000 c.c. of 10 or 20 per cent glucose solution intravenously and from 1000 to 1500 c.c. of saline solution subcutaneously, repeated every eight to twelve hours until a real diuresis of more than 1000 c.c. with a specific gravity of 1.010 or less. Following the injection of fluids we feed the patient through an Andrews' nasal tube, kept in place until she is able to retain food. An outline of this method of treatment which was read before the joint meeting of the St. Louis and Chicago Gynecological Societies in Chicago, Nov. 27, 1927, by Drs. Dieckmann and Crossen, was published in the AMERICAN JOURNAL OF OBSTETRICS AND GYNECOLOGY, July, 1927.

If these patients do not respond, or become definitely worse after six or seven days of this active and intensive treatment, we would consider abortion, but we have not had to do a therapeutic abortion for toxic vomiting of pregnancy since 1921.

Regarding Dr. Titus' paper, I have recently had a private patient deliver spontaneously very shortly after entering the hospital, following which she had two postpartum convulsions. Her blood pressure was 176/110 and her blood sugar was high, 124, although time did not permit our obtaining any blood for chemical analysis before delivery. There was complete dehydration, marked blurring of vision, diagnosed as due to an optic neuritis, and a very scanty urinary secretion. Following the administration of 1000 c.c. of 20 per cent glucose and 1000 c.c. of normal saline solution under the skin, repeated eight hours later, she became apparently normal in every way forty-eight hours after her attack. There was still slight blurring of vision ten days following the attack, though the eyes showed daily improvement. This case seems interesting in that it happened just at the time that Dr. Titus was presenting his paper. Our associate, Dr. W. J. Dieckmann, has corroborated most of Titus' work, and we feel that the need for glucose cannot be overemphasized.

DR. ROBERT D. MUSSEY, Rochester, Minn.—Mann and Higgins have reported recently the results of an experiment which may aid in future investigations of toxemias of pregnancy. They found that the emptying time of the gall bladder is delayed in most pregnant dogs, guinea pigs, and gophers, while gall bladders of normal animals under the same conditions are emptied in normal time. Following a standard meal of egg yolk and cream, the gall bladder of the nonpregnant animal emptied within four hours while that of the pregnant animal did not empty in some cases for several days.

In order to prove that the delayed emptying time of the gall bladder of pregnant animals was not due to increased intraabdominal pressure, the following scheme was devised. Several paraffin balls, sufficient to equal the size of a full-term pregnancy, were placed in the abdomen of a nonpregnant animal. The abdomen was closed and several days later after the animal had recovered from the operation, it was fed the same type of meal. The gall bladders in these animals emptied in normal time.

While this does not have a direct bearing on the subjects of these papers, it will undoubtedly stimulate further investigation of the toxemias of pregnancy. Is the retardation of the emptying time of the gall bladder in pregnant animals asso-

ciated with lowering or abnormality of certain hepatic functions? Is it a part of lowered or disturbed activity of the gastrointestinal tract?

Dr. Kellogg's observation has been borne out by Artz, of St. Louis, who found lowered gastric acidity in pregnant women. We are all familiar with the fact that there is an arrest of the normal emptying time of the stomach during labor, and this may be evidence of a disturbance in the intestinal tract similar to the retardation of the emptying time of the gall bladder. Further investigation of the emptying time of the gall bladder and action of the smooth muscles in pregnancy may be an important supplement to the clinical observations of Dr. Speidel and of Dr. Titus.

DR. E. L. DORSETT, ST. LOUIS, Mo.—In regard to Dr. Speidel's excellent work, I think the majority of us in the past have given too small doses of glucose intravenously. We feel that the initial dose should be much larger than 250 or 300 c.c. intravenously.

With regard to the sudden drops in the blood sugar, that Dr. Titus has so convincingly brought forth, I was wondering if it were possible that this would take place if the patient did not have a convulsion, or, in other words, if we could stop the convulsion, would these sudden drops take place?

We have had 88 cases (all having had convulsions) which we have treated with magnesium sulphate. The results of the first 38 cases, I was delighted with. Our mortality was less than 6 per cent. When we had 60 cases, our mortality went up. When we had had 80 cases, it was a little higher, and then I lost the next three cases. So with 88 cases we have now a mortality of 11 per cent. Even that I think is low, although Williams speaks of 2 per cent and Stroganoff has a much lower mortality but these do not include patients having convulsions.

I believe a very valuable point is the frequent use of glucose. We are prone to give a large dose and then wait for ten or twelve hours before giving another.

DR. WILLARD R. COOKE, GALVESTON, TEXAS.—In a very few cases in which blood-sugar estimations were made, the blood sugar was found to be normal or low. Recently, by accident, two blood analyses were made on one patient, the second specimen being taken just before a convulsion, and it was found that there had been a drop of several milligrams in the blood sugar in the interval of a half hour or so between the taking of the two specimens. We were unable to account for this, and attributed it to error, but it may serve as a corroboration of Doctor Titus' observations.

DR. MILES F. PORTER, FORT WAYNE, IND.—Is there noted any significance in the amount of reaction following the convulsion? That is to say, if the reaction is marked, is that a better sign than when there is a slow reaction? If Dr. Titus' reasoning is correct, it seems to me there should be some significance in the amount of reaction that follows the convulsion.

DR. SPEIDEL (closing).—The essential plea of my paper was to prevent vomiting, as a means of reducing maternal and infant mortality. When it comes to hyperemesis gravidarum, my conception of that condition agrees with that of Dr. Kellogg, that the most important feature of it is an hysterical neurosis and, unless that is controlled, I do not care how much glucose is used you cannot cure the hyperemesis. I challenge any of you to cure a hyperemesis and allow the husband to fondle and pet his wife three times a day, or permit the anxious parents to be in the room.

It is not unusual to find a patient with hyperemesis in the hospital, who has been known to reject even water for a week, and then after perhaps a single injection of glucose and a few doses of bromides to be able to retain fluids. That shows that the hysterical element has been removed, and the patient has returned to normal. My advice is to remove the audience first of all, and you will be

astonished what simple means will restore the equilibrium. I have generally re-sorted to 10 per cent glucose in those cases. I have not used insulin because the patients that I have treated have responded so well to the simple measures. I never inquire as to whether they vomit or not. I get the report of the vomiting from the nurse's chart.

DR. TITUS (closing).—In response to Dr. Porter's question, I must reply that I do not know whether or not there is any prognostic significance to this reaction which we have observed. I think that he is correct in his surmise, but as yet our investigations do not warrant our drawing any such conclusion.

Dr. Dorsett asked if sudden drops in blood sugar still continue to take place after the convulsions are checked. One of our charts shows three cases studied after cessation of convulsions. The fluctuations were not so wide or so sudden, the waves apparently subsiding slowly after the eclamptic storm. The most in-teresting of these three patients was the one whose blood sugar fell fairly rapidly at one point, followed by some twitching which made it appear that a convulsion was imminent. The muscular activity of the twitching was sufficient to send the blood sugar to higher and safer levels, and the expected convulsion did not occur.

I would like very briefly to mention some points in connection with Dr. Speidel's paper. I think he generalizes too much in his treatment; that it should be more specific and somewhat simpler. So far as the injection of 10 per cent glucose is concerned, I have been convinced for some time that this is too weak and have come to this conclusion after having conducted a long period of clinical investiga-tion into this subject, during which I have steadily increased both the amount of glucose given and also the strength of the solution. We know that physiologically a patient can utilize one gram of injected glucose per kilo of body weight per hour. We know that the average woman weighs about 60 kilos; she can therefore take up 60 grams in sixty minutes without spill through the urine. Less than 60 grams would be less than a therapeutic dose, and if less than a therapeutic dose is given, it is as unfair to expect the proper effect from the glucose as it would be to give 1/150 of a grain of morphine and then expect the usual effect merely be-cause it is morphine. The more concentrated the solution the more rapid the inter-change between blood stream and tissues, and the more rapidly the storage of the sugar occurs in the tissues, as a result of which beneficial therapeutic effects are seen more quickly and are more lasting. I am quite insistent upon the point that 25 per cent solution is the proper strength and greatly preferable to the weaker dilutions.

Referring to the work of Dr. Speidel's associate who is giving continuous in-jection, the paper which my associates and I have just presented refers to those clinical experiments which show that a serious hypoglycemia with convulsions and prostration may be produced by glucose injections continued over any considerable period of time. This is caused by the out-pouring of excess insulin from the patient's pancreas in response to such an injection and cannot be combated by the usual antidotes.

This confirms the contention that I have always made, that interrupted injec-tions of glucose frequently repeated are preferable to continuous injection. To add insulin to such injections obviously adds an additional element of danger.

Referring again to our own work as presented today, we venture to make the assertion that these findings lift the intravenous administration of glucose solution for pregnancy toxemias out of the situation of being an empiric measure, and that they establish a definite scientific basis for this treatment which had already proved to be valuable.

DR. JAMES R. BLOSS, Huntington, W. Va., read by invitation a paper on the **Practice of Ideal Obstetrical Technic in the Home.** (For original article see page 424.)

Dr. I. W. Potter, Buffalo, N. Y., read a paper on the **Immediate Repair of Birth Canal Injuries Following Delivery,** With a Lantern Slide Demonstration. (For original article see page 336.)

DISCUSSION

DR. HERMAN E. HAYD, Buffalo, N. Y.—These two papers just presented represent two types of modern obstetrics: the one, the ideal for conservatism of the most thoughtful and the most considerate kind; the other representing the dramatic, the surgical. Both have their advantages.

In the hands of you men who are skilled obstetricians, who are skilled surgeons, Potter's work must make an appeal. The general practitioner who is not acquainted with the possibilities of surgical relief certainly should practice the conservative method.

This question was brought most strongly to my mind by one patient whom I sent to Dr. Potter about ten years ago and to whom he brought two babies. The patient had a badly lacerated cervix, areas of cystic degeneration, and had suffered for years from profuse leucorrhea, and menorrhagia; she was a nuisance to me. She was constantly coming to my office for a number of years. I wanted to operate, and she would not listen to it. Finally she became pregnant; I sent her to Potter and he carried out this procedure which he has shown. A most beautiful involution was the result. Whatever will produce involution is the most desirable in obstetric practice. He also carried out the exercises which our President recommends.

As to the operation of Bubis, who has been doing practically the same kind of work, and has extended it to include the repair of cystocele and rectocele. In the first place, the operation on the anterior vaginal wall for cystocele is very difficult and does not always bring about an ideal result, even under the best conditions. Operations on the perineum are not difficult, and if one brings the levator ani muscle together, a good perineum results, but exposing the woman to the possibilities of infection from without at the introitus it seems to me should contraindicate altogether that line of practice; but what Dr. Potter is doing I would commend.

DR. JAMES E. KING, Buffalo, N. Y.—One feature that has been brought out by this paper, and a feature Dr. Hayd has commented upon, is the great lack of cystocele and anterior vaginal lacerations which we find following Potter's deliveries.

As Dr. Hayd has said, I know of no obstetric birth-canal injury that gives so much difficulty in properly correcting as laceration, or overstretching, of the vesicovaginal fascia, and any method of delivery that lessens the likelihood of such an injury justifies our commendation.

So far as his immediate suture of the cervix is concerned, we should not be surprised that it is coming into prominence at this time. It seems to me rather remarkable that during all these years so little attention has been paid to the freshly lacerated cervix, and I think that we should be pleased that in our day we are giving attention to this important subject.

We must remember that the lacerated perineum of not so many years ago was also disregarded in exactly the same way we have been disregarding the lacerated cervix. In those days the remedy suggested was to bind the limbs together in the hope that it would heal.

DR. D. L. JACKSON, Boston, Mass.—I was taught that secondary repair, that is, denuding an area of the mucous membrane of the perineum and sewing it up, could not be accomplished after labor and that, if it was done, the probability of its holding was very slight. I would like to report

that in the last few years I have seen four cases where a complete tear of the perineum was present. Following a subsequent delivery in each of these cases, I denuded and did attempt to get the ends of the sphincter together and to get support from the lateral muscles with a completely satisfactory result in each instance. The only difficulty, I think, to be encountered is due to bleeding from the extensive venous supply.

DR. POTTER (closing).—This work can be done in from three to five minutes, and it does not jeopardize the condition of the patient at all. Perhaps I have made too many of these repairs. In my enthusiasm I have done it routinely. I wanted first to perfect a technic, and I wanted to see the results. I do not think it is open to criticism at all to say that this thing should be done routinely.

DR. LOUIS E. PHANEUF, Boston, Mass., read a paper on **Vaginal Cesarean Section**. (For original article see page 325.)

DR. ROBERT D. MUSSEY, Rochester, Minn., read a paper on the **Classification of Nephritis in Relation to the Prognosis for Pregnancy**. (For original article see page 366.)

DR. WALTER T. DANNREUTHER, New York, N. Y., read a paper entitled **The Prophylaxis of Postoperative Pyelitis**. (For original article see page 406.)

DISCUSSION

DR. LOUIS E. PHANEUF, Boston, Mass.—Dr. Dannreuther's incidence of pyelitis following operation was much lower than mine. If I reviewed 500 gynecologic cases I would find more than 2.5 per cent. I have felt that plastic operations predispose to this condition, but that is evidently not substantiated by Dr. Dannreuther.

DR. G. D. ROYSTON, St. Louis, Mo.—The phthalein test and blood chemistry have been disappointing at times, and in the final analysis of certain obscure cases we have had to depend almost solely upon the clinical findings. Those conditions that are very definitely nephritic offer no difficulties, but it is the obscure case that is puzzling. At such times we have depended almost solely upon two things: the steadily increasing blood pressure, and the twenty-four-hour output of urine.

DR. F. A. CLELAND, Toronto, Ont.—I do not know what my incidence of postoperative pyelitis is, but I know that we have not been bothered nearly so much lately as formerly. I think the reason is that we have, to a large extent, developed a prophylaxis as suggested by Dr. Dannreuther.

Even with our public ward cases we conduct our clinics so that we operate only three days a week, and the patients must be in the hospital the day before operation for a clinic to the students. In that way we get the patients in the hospital earlier. We do not allow them to come in at nine or ten o'clock at night for operation the next morning. That is a pernicious habit, and I find it still persists in some hospitals.

We also carry out a postoperative treatment that I think is very beneficial. The nurse is instructed to keep the abdominal patient warm after operation. She must have a large linseed poultice placed over the abdomen; that is done automatically. If the patient requires it, we give her a subcutaneous injection of normal saline solution, but the important instruction is that as soon as the patient can be induced to drink hot water she must do so. If she will take it with soda bicarbonate in the water, well and good. If that is objected to, she must be forced to take hot water.

As far as I am personally concerned, in making my rounds postoperatively, I am very much more interested to see how much urine the patient has excreted than to see whether the bowels have moved. I think that is a very important point in prevention of pyelitis postoperatively.

DR. JAMES E. DAVIS, ANN ARBOR, MICH.—May I call attention to a peculiar ability the kidney possesses under stress and strain in its reaction to injury that is perhaps not possessed by any other organ in the body. It is indeed very rare to see kidneys diffusely involved, excepting in very acute conditions. If one will follow cases of acute nephritis through to the end picture, he will be perfectly amazed to find that the picture has changed from a condition in the acute stage of apparent entire involvement of both kidneys to focal degenerative changes; or, if examined in the period before the terminal event, there will be focal inflammatory conditions.

The term focal nephritis is used loosely, and ordinarily one expects to find very definite abscesses in the kidney before he is willing to say it is a classical case of focal nephritis. This is too exacting a requirement, for in many instances no abscesses are formed; only aggregations of lymphocytes are observed.

Just in this connection may I call attention to a biologic feature in connection with the kidney? Richards of Philadelphia, I believe, was one of the first to place our knowledge of the alternating and rhythmic glomerular function upon a very sound scientific basis. He was able to observe positively, and to have others see what he saw; namely, the nephron units of the kidneys actually functioning, and it was a great surprise when he first observed that the units did not act all together or in unison. So one may consider all the two million units or nephrons of the kidney and find that only a certain percentage of the entire group are at work at any one given time. It is logical, then, to assume that similar changes in regard to the ultimate pathologic results; namely, that one part of the kidney will have a different kind of response from other parts, and the parts of the kidney that are definitely weak will be the first to go down under the attacks of acute nephritis, or chronic nephritis, or the repeated nephrites. So the pictures at different periods of life vary so widely that it is a most confusing thing to postulate accurately the end-results.

DR. MUSSEY (closing).—I wish to emphasize the point made by Dr. Royston, that the chemical examinations of the blood which we have at our disposal do not show any changes in the blood in cases of acute nephritis but that in chronic nephritis these examinations are frequently of a great deal of importance.

In the discussion of Dr. Dannreuther's paper, the question was raised of pyelitis following confinement. Medical students of twenty or more years ago were taught that catheterization of the parturient woman was a dangerous procedure. We now know that the pregnant woman and parturient woman may be catheterized under aseptic conditions without danger. Several years ago we catheterized the bladders of 200 consecutive postpartum patients. In each instance, the patient was catheterized after she had voided for the first time or if she did not void within eight hours. In over half the cases, residual urine was found in the bladder after the patient had voided, and the retention was greater in those patients who had difficult instrumental deliveries. In a good many instances, pus was found in the centrifuged specimens. While this observation is now new, it explains some of the cases of fever which occur postpartum and which may be avoided by not allowing residual urine to remain in the bladder.

DR. DANNREUTHER (closing).—Referring to Dr. Pantzer's remarks first, I may say that unfortunately there are no "suspicious" cases. My experience has been that this type of pyelitis develops unexpectedly between the tenth and eighteenth postoperative day. All of us must admit that pyelitis is observed far more often during pregnancy than as a postoperative complication, and I also

venture the assertion that chronic pyelitis is much more common in office practice than is generally supposed, unless cystoscopic investigations are made in all patients with lumbar discomfort or urinary symptoms.

In reply to Dr. Polak's inquiry concerning the care of the bladder in the postoperative period, I can truthfully say that I believe that more cases of cystitis are due to a failure to use the catheter sufficiently often than occur because of its use. I have a standing order that all patients shall be catheterized every six hours for three days, irrespective of voiding, after hysterectomy and after plastic operations (except fistula cases). Residual urine constitutes much more of a menace than does instrumentation. In cases of vesicovaginal or urethrovaginal fistula, I leave a Pezzer retention catheter in the bladder for two weeks, instilling a little weak silver nitrate solution through it daily, and the results have been gratifying. It is advisable to prescribe hexamethylenamin and acid sodium phosphate early in these cases as a prophylactic measure. Hexyl-resorcinol is contraindicated in these cases too, because its use involves the restriction of fluid intake to maintain the surface tension of the urine, which is of course a serious handicap to postoperative patients.

The presence of pus cells in the urine is significant and of diagnostic importance, but I believe that a culture of the urinary sediment is a more reliable index of infection. I desire to emphasize, however, that the discovery of the colon bacillus in the specimens from all six of the patients occurring in my series of 500 cases is no criterion that other microorganisms, such as the staphylococcus and streptococcus, may not cause the same mischief.

A word of warning should be uttered regarding the routine use of bicarbonate of soda. I was formerly so enthusiastic about it, to protect the patient against the precipitation of a postoperative acidosis, that I not only gave the patient this drug for several days before the operation, but also used it in a Murphy drip after operation. A few years ago, however, three postoperative patients at the New York Post-Graduate Hospital died of alkalosis. In two cases alkalis had been used as a matter of routine. The third patient was my own, and the only reason that I escaped responsibility was because she was an emergency case. I had no opportunity to give preliminary therapy and was concerned chiefly with transfusions after operation. She died on the eighth day from alkalosis with an incidental high blood urea nitrogen. Ever since then I have refrained from the use of alkalis as a matter of routine, unless I know what the carbon dioxide combining power of the blood is beforehand.

DR. DAVIS (closing).—There are a number of different hematurias, one caused by turpentine, another from hydrostatic conditions, others from anemic conditions, and some from general vascular disease, especially if it is in the very early or very late periods of life. In an examination of the kidney in hematurias, one finds only a slight bluish-red appearance macroscopically in the kidney, just suggesting a slight degree of passive congestion. Microscopically, it is surprising to find that there is often no structural change. If called upon to make a microscopic diagnosis, one would say the kidney is normal. There is a clinical history of bleeding for months and one feels he must have missed the involved area. I have gone over a number of kidneys of this type to find the bleeding area, but usually without success.

In interstitial nephritis, hemorrhages may take place in various parts of the body, and not infrequently in nasal and gastrointestinal mucosal surfaces. I know of no explanation, excepting that the smaller vessels break and bleed slightly, then close up so that without almost immediate examination the damage in the vessel cannot be recognized.

When patients have died in a few days following rather simple operations, and the anesthetic has not been prolonged, and there has been very little trauma

with but little bleeding at the time of operation, careful examination at the autopsy table will show that these patients have a multiple number of small hemorrhages in different parts of the body, with a marked interstitial type of change in the kidney, liver, pancreas, and heart. Examination of the vascular system will show it is in a degenerative condition.

DR. FOSTER S. KELLOGG, Boston, Mass., read a paper on **Premature Separation of the Normally Implanted Placenta With Special Reference to the Kidney in These Cases**. (For original article see page 357.)

DISCUSSION

DR. PAUL TITUS, PITTSBURGH, PA.—One of the most important points brought out by Dr. Kellogg is that increased blood pressure is probably a protective measure. We should be exceedingly cautious about overbleeding the ordinary toxemia patient for fear we might destroy this very protective measure.

There is no question that chronic nephritis has a definite association with these conditions, but is it not likely that chronic nephritis adds merely a general susceptibility rather than that chronic nephritis is always to be found in a toxemia of pregnancy? It is not always associated with it, nor are all of these cases accompanied by even an acute nephritis. That would tend to confirm the idea that the acute nephritis we see so often in toxemia is a secondary matter rather than a primary one.

Dr. Kellogg pointed out in his chart that the blood chemistry of this patient was normal except for a lowered blood sugar. This I would expect and consider an important fact. May I suggest that following intravenous glucose injections, spill of sugar through the urine is not so much dependent upon the height to which this injection sends the blood sugar as it is upon the rapidity with which the glucose injections are given.

Many times accidental separations of the placenta are seen without toxemia and without nephritis. This is a complex problem beyond doubt, and I believe it must be agreed that in one instance we may have a certain set of concomitant clinical symptoms, and in another instance another set. The observation on blood pressure impresses me as one of the most valuable points that Dr. Kellogg has made in this excellent work.

DR. ROBERT D. MUSSEY, ROCHESTER, MINN.—I would like to ask the essayist concerning the presence of edema in the anuric cases. In our waterlogged cases or in those patients with a marked amount of edema and oliguria or anuria, the fluid intake is kept down until the patient begins to excrete more urine. An effort has been made to dislodge the increased retention of sodium chloride in the cell by using either ammonium chloride or ammonium nitrate. Following the intake of ammonium, there is a freeing of sodium chloride which is held in the cell and which is apparently associated with the retention of water. This is usually followed by the free output of urine. Coincident with this, the edema disappears and the patient's condition improves. Then more fluids are given. If edema is not evident, there is no indication for the restriction of fluid intake.

DR. JAMES E. DAVIS, ANN ARBOR, MICH.—Something might be said about congenital syphilis as a cause, and about developmental conditions. Where the placenta shows a cord attached eccentrically, the plexus of vessels is inadequate; that is, the plexus will spread out with the trunks attached marginally with the main attachment of the cord. This is a developmental condition that always carries a liability. I believe every placenta should be examined to determine the conditions in the chorion laevic zone, chorion frondosic zone, the cord and its position of attachment, the chorionic and amniotic membranes, and the cotyledons.

The chorion laevie zone may be unusually and relatively thickened, ridged, and broadened so as to diminish the frondosic zone. Extreme eccentric cord attachment is always a hazard for the placental circulation. The fusing of cotyledons and the irregularities in their sizes together with their vascularity are usually indicative of important pathologic changes. The gross examination of the placenta will amply repay for the short time it requires, and it will enable the selection of the most promising sections for microscopic examinations.

DR. KELLOGG (closing).—Having spent the last few months looking at these cases, I feel that I do not know much yet about this phase of the subject, and my only solace is that I do not believe anybody else does.

I purposely avoided the question of etiology in order to make the paper brief and to consider only the kidney group. I am firmly convinced, however, that Dr. Polak's contribution regarding excessive right rotation is a real etiology in some cases. I have cut into two uteri at a single mid-line stroke to try to get a live baby and cut into the cornual end of the left tube. I think there is usually a certain amount of very mild trauma connected with these right rotation cases.

Going back to Dr. Davis' question, we need badly a laboratory devoted to the study of obstetric pathology and a far higher percentage of autopsies.

Regarding Dr. Mussey's question about edema, we have not made the accurate observations that he has made in the study of edema, but we assume that edema fluid is not available as a kidney diuretic. Some of these patients were edematous and some were not. Almost all showed at least a little. We always treat our eclamptic and toxemic patients, whether with premature separation or not, as though they were dry, and our results have been better I think since we have saturated them with fluids. This is a point, it seems to me, often overlooked in treatment; most patients have some fluids, many not enough.

The kidney in relation to separated placenta, to my mind, is almost untouched with a reasonable amount of study. J. Whitridge Williams makes two observations in his textbook about it which has especially interested us. He says, in the first place, that he has seen "so many" cases but only one that was a chronic nephritic. This statement is pretty well in accord with our observations so far as we have gone and can prove; yet we *believe* strongly that further and more intensive study over a period of time will show a group not negligible associated with chronic nephritis. This textbook says that the etiologic factor is presumably of the nature of the etiologic factor in toxemia, but not the same. Ultimately this statement may or may not prove to be true. As evidence against it we have stressed eight cases in which placental separation took place coincident with an actual toxemia of pregnancy. These cases we offer as some evidence that in one group at least the etiologic factor of the two conditions is the same.

Books Received

TEXTBOOK OF PRACTICAL THERAPEUTICS. By Hobart Amory Hare, Professor of Therapeutics, etc., Jefferson Medical College, etc. Twentieth edition, enlarged, thoroughly revised and largely rewritten. Lea and Febiger, Philadelphia, 1927.

GYNECOLOGY FOR NURSES. By Harry Sturgeon Crossen, Professor of Clinical Gynecology, Washington University Medical School, etc. With 365 engravings, including one color plate. St. Louis, C. V. Mosby Company, 1927.

GLASGOW ROYAL MATERNITY AND WOMEN'S HOSPITAL. Medical report for the year 1926. William Hodge and Co., Lim., Glasgow, 1927.